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EMBOLECTOMY FROM THE ABDOMINAL AORTA*

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BOSTON

ONE hundred and ninety saddle emboli at the aortic bifurcation have been reported to date. To these we are adding 3 cases, making a total of 193. Two of our 3 patients were treated by embolectomy, and 1 by supportive measures, including lumbar sympathectomy without removal of the embolus. Amputation of a leg was required in 1 case, in which embolectomy was performed thirty hours after onset. The circulation of the leg was restored in the other cases — 1 with and 1 without removal of the embolus.

Undoubtedly, many more aortic emboli have occurred than have been reported. In several reports[1-3] aortic thrombosis has been confused with aortic embolism. Of the 193 patients, 144 died as a result of the embolus (Table 1). Twenty-six patients were cured by direct or indirect embolectomy. Eleven patients survived after amputation. Eight patients were cured by conservative measures short of removal of the embolus, and 4 cases were reported as "spontaneous cures without any treatment."

The term "cure" is considered to mean complete restoration of circulation to both legs for a period of at least three weeks after operation with no loss of tissue as a result of the disturbed circulation. The 4 spontaneous cures without any treatment may not represent aortic bifurcation embolism but rather a phenomenon of vasospasm, as reported by Atlas[32] in 1942. His patient showed complete obliteration of all leg pulsations bilaterally, including the femoral arteries, that was due to reflex vasospasm associated with a unilateral peripheral arterial embolus.

Conservative treatment includes all therapy other than embolectomy — namely, the vasodilator drugs, the anticoagulants heparin and Dicumarol, sympathetic-nerve interruption, Buerger's exercises, intermittent venous occlusion and cooling of the extremities to lower their metabolism. These measures may prove extremely important as ancillary treatments after the embolectomy. In our third patient bilateral lumbar sympathectomy was combined with removal of the embolus.

It is apparent from Table 1 that most patients with embolism at the aortic bifurcation die, with or without amputation, unless the embolus is removed (there were 144 deaths in 193 cases). These patients must survive not only a major vascular catastrophe but also their underlying cardiac condition, which is often serious, and frequently other embolic phenomena as well.

The twenty-six successful aortic embolectomies (Table 2) are considered to comprise a complete accounting of the cases to date. These were all of cardiac origin. In Madden's[3] recently published case the patient died three months after success-

TABLE 1. *Results in 193 Cases of Embolism at the Aortic Bifurcation.**

AUTHOR	DATE	RESULT			
		SURVIVALS AFTER OPERATION	SPONTANEOUS CURES WITHOUT TREATMENT	CURES BY CONSERVATIVE TREATMENT	CURES BY EMBOLECTOMY†
Welch and Rolleston[4]	1909	—	—	3	—
Bauer[5]	1913	—	—	—	1
Hesse[6]	1921	2	2	—	—
Key[7]	1922	—	—	—	1
Gamble[8]	1930	—	—	—	1
Walzel[9]	1930	—	—	—	1
Dreverman[10]	1931	—	—	—	1
Usadel[11]	1931	—	—	—	1
Neuhof[12]	1932	—	1	—	—
Nyström[13]	1932	—	—	—	1
Westerborn[14]	1932	—	—	—	1
Gohrbandt[15]	1933	—	—	—	1
Schmorrell[16, 17]	1933	—	—	—	1
Stegemann[18]	1933	—	—	—	1
Lund[19]	1937	—	—	—	1
Culmone[20]	1938	—	—	—	1
Jefferson[21]	1938	—	—	—	1
Lindgren[22]	1939	—	—	—	1
Ravdin and Wood[23]	1941	—	—	—	1
Herrmann[1]	1943	—	—	1	—
Murray[24]	1943	—	—	—	4
Reich[25]	1943	—	—	1	—
Wikle and Cabot[26]	1943	1	—	—	—
Linton[27]	1945	—	—	—	1
Cooper, Harris and Kahn[28]	1948	—	—	—	1
Holden[29]	1948	—	—	—	1
Morest and Rubin[30]	1948	1	—	—	—
Warren and Linton[31]	1948	6	1	2	1
Madden[3]	1949	—	—	—	1
Albright and Leonard	1949	1	—	1	1

*Of the total number of cases, 144 were immediately fatal.
†The cases in this column are further analyzed in Table 2.

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ful aortic embolectomy, and 9 days after unsuccessful resection of the left auricular appendage of the heart. The patient in the case reported below, at sixty-five years of age, is the third oldest (sixty-eight years, sixty-six years, and sixty-five years, respectively) on record to have had a curative operative result. This embolectomy was done by direct opening of the abdominal aorta. Only 7 previously reported cases have been successfully treated by the embolus around over the aortic bifurcation to emerge through a single opening in the right common iliac artery. Transabdominal, retroperitoneal and femoral approaches have been used. Distal tourniquets have been employed with these retrograde methods to prevent distal propulsion of fragmentation emboli during the maneuvering of the saddle embolus. Such use of distal tourniquets in the case reported below might have fragmented the long,

TABLE 2. *Data in 26 Successful Aortic Embolectomies.*

AUTHOR	DURATION OF EMBOLISM	SEX	AGE	END RESULT
	hr.		*yr.*	
Direct aortotomy:				
Bauer[5]	.3	M	39	Death 2½ yr. after operation
Walzel[9]	1	M	38	Death 25 days after operation
Dreverman[10]	6	M	41	Patient living at 1 yr.
Usadel[11]	½	F	44	Patient living at 9 mo.
Gohrbandt[15]	5	F	39	Unknown
Lindgren[23]	10	F	41	Unknown (patient living at 9 days)
Madden[3]	5	M	52	Death 3 mo. after operation
Albright and Leonard	1½	F	65	Patient living 10 mo. after operation
Aortectomy:				
Cooper, Harris and Kahn[28]	(5 mo.)	M	22	Patient living at 5 mo.
Retrograde milking:*				
Key[7]	23	M	44	Death 10½ yr. after operation
Gamble[8]	2	M	68	Death 3 wk. after operation
Nyström[13]	½	M†	56†	Recurrence at 9 mo.†
Westerborn[14]	6	M†	56†	Death 5 yr. after operation†
Schmorell[16, 17]	—	M	66	Patient living at 5 wk.
Stegemann[18]	4	F	55	Death after several mo.
Lund[19]	9	F	34	Patient living at 5½ yr.
Jefferson[21]	6	M	25	Death 3 mo. after operation
Culmone[20]	4	F	50	Patient living at 3 mo.
Ravdin and Wood[23]	3	M	32	Patient living at 3 yr.
Murray[24]	—	—	19	Death 2½ yr. after operation
Murray[24]	21	F	38	Death 4 mo. after operation
Murray[24]	—	F	48	Death 2 yr. after operation
Murray[24]	—	F	27	Patient living at 3½ yr.
Linton[27]	3	F	39	Patient living at 16 mo.
Holden[29]	1½	F	58	Death 12 days after operation (autopsy showed coronary occlusion, with aorta free of embolism)
Warren and Linton[31]	8	M	56	Death 3 mo. after operation

*By some form of unilateral or bilateral iliac or femoral approach.
†Same patient.

this approach (Table 2). The reasons given for avoidance of opening into the aorta have been the danger of its mobilization and control. The youngest patient was nineteen years of age.

The case of aortectomy reported by Cooper et al.[28] was unusual. A young soldier had been shot in the chest. The 0.45-caliber bullet was found to be lodged within the left ventricle, whence it migrated to the aortic bifurcation, and here it was observed for five months. Subsequent aortitis, aortic-wall erosion and local progressive thrombosis made it necessary to remove not only the bullet but also the entire bifurcation. Adequate collateral circulation had become established, and the soldier's leg circulation was reported actually improved after operation.

Table 2 shows the preponderance of successes by some form of retrograde maneuvering of the embolus from below. Various methods have been employed. Ravdin and Wood[23] used catheters put up from each opened femoral artery. Linton[27] milked 15-cm. tail clots extending bilaterally into each iliac and femoral artery.

In all but 5 cases in Table 2 the patients were operated upon within six hours of the embolism,

TABLE 3. *Sites of Lodgment of Peripheral Arterial Emboli.*

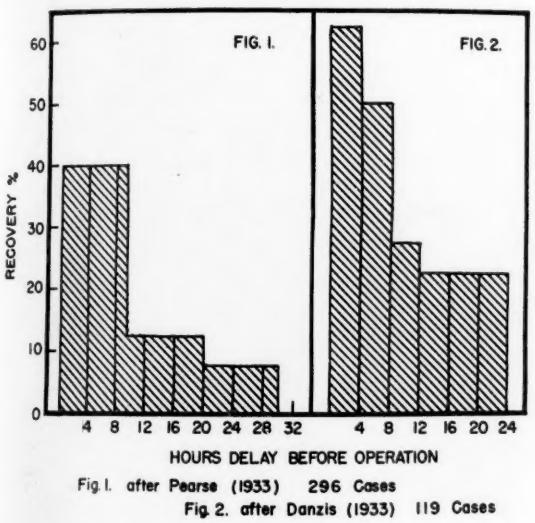
SITE	INCIDENCE
	%
Femoral artery	54.4
Iliac artery	17.2
Axillary artery	11.8
Brachial artery	11.8
Popliteal artery	11.3
Aorta	4.5*
Carotid artery	—
Subclavian artery	—

*8.7 per cent (15 of 172 patients) at the Massachusetts General Hospital, 1937-1946. The frequency of aortic saddle embolism of the bifurcation ranges between 4 and 8 per cent of all peripheral arterial emboli.

excluding the unusual aortic segmental resection employed by Cooper and his associates.[28] This emphasizes the desirability of early intervention. Isolated cases of cure after delay up to twenty-

three hours (Key[7] and Murray[24]) suggest that patients with late cases should not be denied embolectomy because of elapsed time alone. Nyström[13] and Westerborn[14] operated separately on the same patient, who survived two successful aortic embolectomies and lived for five years. Schmorell's[16] patient was operated on during a generalized peritonitis of appendiceal origin, with successful outcome by a retroperitoneal approach.

The relation of time elapsed between peripheral embolism and embolectomy as related to survival is shown in Figure 1 and Figure 2. Six to eight hours' delay appears to be the upper limit permissible for best chances of survival of the limb. Among the 26 successful aortic embolectomies (Table 2) the delay was recorded in 21 cases. In 16, or 76 per cent, of the successful cases operation was performed within six hours. Nineteen patients were operated on within ten hours, and the remaining 2 at twenty-one and twenty-three hours respectively. Certainly, successful outcome seems to be related to early intervention — namely, within six hours — in the face of the discouraging prostration of the patient.



FIGURES 1 AND 2. *Relation of Time to Recovery in 415 Cases of Peripheral Arterial Emboli (Reproduced from Hopkins[33] with the Permission of the Publishers).*

Both figures show that the rate of recovery ranges between 40 and 60 per cent if the patient is operated on within the first eight hours. The rate drops sharply with delay beyond eight hours.

Therapeutic trial of novocain block of the lumbar sympathetic nerves and anticoagulants during the first six to ten hours after aortic embolism has been suggested.[34, 35] These should in our opinion form the basis of conservative treatment when operation has been decided against because of the grave condition of the patient. Conservative treatment alone led to survival in 11 of 193 patients, or 5.6 per cent (Table 1). In many cases the initial prostration appeared to be due to primary shock that was quickly responsive to morphine. This was evident in both our cases in which operation was done, with quick return of the blood pressure to levels of 150 systolic, 80 diastolic. Conservative measures, especially lumbar novocain blocks, heparin and Dicumarol, may best be combined with or follow embolectomy, but in our opinion should not be allowed to postpone operation.

Objection has properly been raised to the use of lumbar sympathetic novocain blocks on patients under anticoagulant influence.[36] Large and sometimes disastrous retroperitoneal hematomas have been encountered. Excision of the lumbar sympathetic nerves bilaterally at the time of embolectomy, as carried out in our second patient, operated on by Dr. Thomas J. Anglem, would obviate this objection. In this forty-six-year-old patient an aortic embolus was removed transabdominally by Linton's retrograde technic, with removal of segments of the lumbar sympathetic nerves bilaterally at the same time. The embolism had occurred twenty-four hours before the patient was seen, and irreversible changes in the left leg before embolectomy necessitated subsequent high amputation of the left thigh.

Peripheral arterial emboli (Table 3) tend to lodge at the aortic bifurcation in 4.5 to 8.7 per cent of the cases. The heart with auricular fibrillation or myocardial infarction (Table 4) was the major source of 172 arterial emboli in 98 patients seen at the Massachusetts General Hospital from 1937 to 1946 (Warren and Linton[31]). In rare cases syphilitic aortitis, aortic aneurysm and atheroma, intracardiac thrombosis in the congested heart and pulmonary-vein thrombosis have led to embolus. Thrombosis in a closing ductus arteriosus (Gross[37]), vegetations on an anomalous heart valve, spontaneous clotting in the polycythemia of "blue babies," myxoma fragments from a myocardial tumor (Young and Hunter[38]) and a migratory bullet (Cooper[28]) have given rise to emboli.

"Paradoxical emboli" were first described by Cohnheim[39] in 1877, his case being associated with

TABLE 4. *Origin of 172 Arterial Emboli to All Sites in 98 Patients.**

CAUSE	No. OF CASES
Auricular fibrillation	64
Myocardial infarction	15
Subacute bacterial endocarditis	7
Auricular fibrillation and myocardial infarction	1
Undetermined	11
Total	98

*Massachusetts General Hospital Series, 1937-1946 (Warren and Linton[31]). In 80 of these patients the emboli arose in the heart damaged by auricular fibrillation or myocardial infarction, or both.

a patent interauricular foramen ovale. Other paradoxical emboli from the venous circulation have transversed an interventricular septal defect (Birch[40]).

Case Report

L. F., a 65-year-old single woman, was admitted to the Sancta Maria Hospital on November 12, 1948, with an embolism of the left popliteal region of 6 days' duration. She had been digitalized for 6 years for known rheumatic and arteriosclerotic heart disease with mitral stenosis and auricular fibrillation. There had been no decompensation. Two previous emboli, one to the left leg 2 years previously and one to the right leg 1 year previously, had occurred, without disability.

The immediate cause of admission was a third-degree burn of the entire left calf and heel (Fig. 3), the result of treatment by an electric heating pad adjusted to the "low" position for 3 days. This burn was found at later operation to have penetrated 5 cm. through the muscles of the calf and also to involve the os calcis. The ischemic leg had been kept elevated. The pulsations were absent from the left popliteal artery distally.

Heparin was given intravenously in 100-mg. and 75-mg. doses, in addition to 300 mg. of Dicumarol orally, to the digitalized, slowly fibrillating patient, and a left lumbar sympathetic block with novocain was carried out

Figure 3. *Appearance of the Charred Burn of the Left Calf, Underlying Muscles and Heel on Admission. An electric pad had been used in the treatment of the left popliteal embolism.*

On the 2nd day, after 300 mg. of Dicumarol and while the fourth dose of heparin was being administered, severe pain in both legs, profuse sweating and alarming prostration occurred. The blood pressure was 120/80, and a dramatic line of demarcation was noted across the lower abdomen (Fig. 4), below which the skin was of a cadaveric, mottled-marble hue. Both femoral pulses and all distal pulsations were absent. The patient could but weakly move her toes. Morphine in a dose of 10 mg. (1/6 gr.) was given, and preparations were made for embolectomy which was performed 1½ hours later. The blood pressure rose to 150/80 before operation.

Under nitrous oxide, oxygen and ether anesthesia, through a long left paramedian incision (Fig. 4), the coils of intestine were retracted upward and to the right, with ready exposure of the aorta. Vigorous pulsations ended abruptly at a firm, rounded 3.5-cm. mass at the bifurcation. The aorta was separated from its bed on the vertebral bodies and from the vena cava. To avoid injury to the lumbar arteries, the aorta was raised only enough to allow passage of a soft-rubber Penrose tubing beneath it, proximal to the clot. The tubing was then twisted down on the aorta (Fig. 5) only enough to occlude the aorta without damage to its wall.

A 3.5-cm. incision was made longitudinally through the anterior aortic wall, directly over the embolus, which was

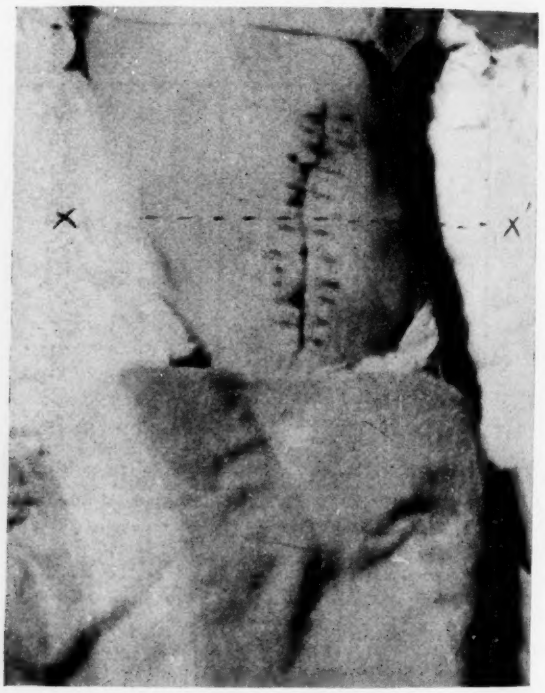


Figure 4. *Healed Incision, Fourteen Days after Operation. The line X—X' indicates the line of demarcation with distal mottling and purplish discoloration before embolectomy. The line receded rapidly after removal of the embolus and disappeared from the lower legs on the following day.*

removed in one piece. Catheter suction was applied distally to the iliofemoral arteries bilaterally, with removal of currant-jelly tail clots, 15 cm. long. Distal tourniquets were purposely avoided so as to allow as complete suction removal of the distal clots as possible, and indeed no need for control of distal hemorrhage arose.

The aortic opening was then closed with two interdigitating layers of interrupted fine-silk sutures. The tourniquet was removed, gelfoam was maintained in position over the suture line for 5 minutes by manual pressure, and the abdomen was closed with through-and-through silk sutures. The line of demarcation by this time had receded quickly to midthigh level, and bilateral femoral pulsations were strongly evident. The demarcation had completely disappeared on the following day.

Postoperatively, heparin and Dicumarol were continued, and bilateral lumbar sympathetic blocks with novocain were performed as outlined in Figure 6. Normal pulsations were ultimately restored in the right leg and in the left leg down to the popliteal artery, but the right, unburned leg caused more concern in the early period. One hundred per cent oxygen therapy with a BLB mask was given to maintain

maximum oxygen content especially of the peripheral blood. Subsequent débridements (D & D in Figure 6) of the left-calf burn necessitated removal of the gastrocnemius, soleus

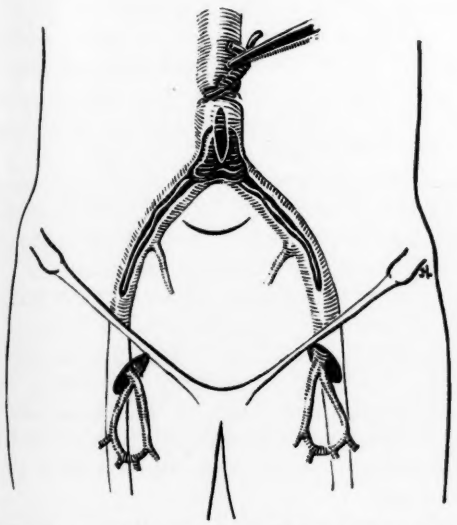


FIGURE 5. *Approach to the "Saddle" Embolus, with Complete Control of Bleeding by the Twisted Soft-Rubber Tubing and no Distal Tourniquets.*

Note the clot extending into both iliac arteries.

and plantaris muscles down to the pulsating posterior tibial artery and the plantar flexor muscles of the foot (Fig. 7).

Because of the severe loss of tissue from the left calf and heel, incurred by the burn 6 days before the bifurcation em-

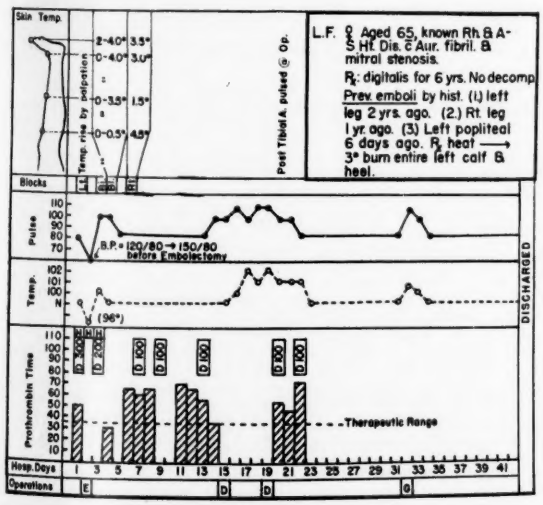

Skin Temp.
Temp. rise by palpation
Post Tibial A. pulsed @ Op.
L.F. ♀ Aged 65, known Rh & A-S Ht. Dis. & Aur. fibril. & mitral stenosis.
℞: digitalis for 6 yrs. No decomp.
Prev. emboli by hist. (1.) left leg 2 yrs. ago. (2.) Rt. leg 1 yr. ago. (3) Left popliteal 6 days ago. ℞ heat ⟶ 3° burn entire left calf & heel.
Blocks
Pulse
B.P. = 120/80 → 150/80 before Embolectomy
Temp.
(96°)
DISCHARGED
Prothrombin Time
Therapeutic Range
Hosp. Days
Operations

FIGURE 6. *Summary of the Patient's Postoperative Course and Treatment. (Anticoagulants Were Discontinued on the Twenty-Second Day, and the Patient Was Discharged on the Forty-Second Postoperative Day.)*

E = *Embolectomy*; D = *Débridement*; G = *Skin grafting.*

bolism, it was believed that the success of the outcome was firmly established by the restoration of circulation in both extremities. Not only was pulsation of the left posterior tibial artery present but also the circulation was adequate to sustain a 100 per cent take of the skin graft to the entire left calf on the 32nd postoperative day. The graft over the exposed os calcis failed to take. Flexion of the plantar muscles and of the undisturbed extensor muscles of the left foot returned to a limited degree. The patient was discharged on the 42nd postoperative day, using crutches but in a weakened state, which was due chiefly to her severe cardiac status. Her condition has remained essentially unchanged for 11 months. No further emboli have occurred. A recent photograph (Fig. 8) shows part of the grafted left calf. The only

FIGURE 7. *Completed Débridement on the Nineteenth Postoperative Day.*

The gastrocnemius, soleus and plantaris muscles were removed. The posterior tibial nerve is shown coursing over the probe. The posterior tibial artery pulsated. Grafting of this defect on the thirty-second day was completely successful except over the exposed os calcis.

defect is the persistent draining point, 1 cm. in diameter, overlying a localized chronic osteomyelitis in the os calcis. This is the occasion for the ankle bandage in the photograph. The patient is up and about on crutches, but has occasional bouts of cardiac decompensation with pulmonary edema.

DISCUSSION

Several important points are illustrated in this patient. Diagnostically the "five P's" of arterial embolism were present: pain, pallor, paralysis, pulselessness and paresthesia. Therapeutically two "don't's" in arterial embolism were demonstrated: avoidance of elevation of the limb and of local application of heat. Depression of the ischemic limb allows the diminished blood supply entering it to remain longer. Locally applied heat, by increasing the local tissue metabolism, resulted in a third-degree burn in this patient even though the electric pad was on the "low" position for three days.

That embolectomy was performed on this patient within one and a half hours of the acute episode of embolism is worthy of comment. It should also be emphasized that the patient had already received heparin and Dicumarol, and was actually receiving heparin intravenously when the embolism occurred. The salutary response of the vascular collapse after a single dose of morphine in the early

period added considerably to our hope for a successful outcome with embolectomy.

The direct approach to the aorta in this patient, aided by a proximal soft-rubber tourniquet, in the absence of desirable Bethune vascular clamps, enabled direct control of the embolus, with no troublesome bleeding and removal of the extensions of the clot. Had distal tourniquets or clamps been applied on each iliac artery and the contained clot, fragmentation might have occurred, with possible peripheral dissemination if the fragments were not completely removed. The soft-rubber tourniquet recommended itself here over more special devices only because of its availability and simplicity, and because it worked.

FIGURE 8. *Most Recent Photograph of the Patient Taken Ten Months after Embolectomy (the Picture Also Shows the Completed Skin Grafting).*

The bandage on the ankle covers a small, draining 1-cm. defect in the os calcis, which is the site of a draining localized chronic osteomyelitis.

SUMMARY

Three cases of saddle embolism of the aortic bifurcation are added to existing reports, making a total of 193 cases found in the literature. Twenty-five previously reported successful aortic embolectomies are reviewed to which another successful case is added and described in detail. Direct aortotomy, with a simple rubber tourniquet applied proximally, is discussed as a method of embolectomy. Early removal of the embolus is recommended, to be followed by conservative ancillary measures designed to promote the peripheral circulation.

Experience indicates that embolectomy is the method of choice in the treatment of saddle embolus, provided the patient is not in irreversible shock. In the 193 cases recorded to date there were 8 cures by conservative treatment and 26 cures by embolectomy. The remaining patients died regardless of the treatment.

Conservative measures, especially anticoagulants and lumbar sympathetic blocks, may be combined with or may follow — but in our opinion should not be allowed to postpone — the operation.

Best results have followed embolectomy performed within six hours of the embolism.

Embolectomy should not be denied a patient on the basis of elapsed time alone. The longest delays, with successful outcome, were twenty-one and twenty-three hours respectively. The oldest patient was sixty-eight years, and the youngest nineteen years of age.

Direct aortotomy, with the use of a soft-rubber tubing twisted as a temporary proximal tourniquet and with no distal clamps, kept the procedure simple, allowed suction removal of the distal secondary clots and led to a successful outcome in the case reported.

Transabdominal bilateral lumbar sympathectomy at the time of embolectomy may prove advantageous when the operating time is not unduly prolonged and the patient's condition permits.

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A LOW-SODIUM DIET FOR HYPERTENSIVE VASCULAR DISEASE PRECIPITATING ADDISON'S DISEASE AND MILIARY TUBERCULOSIS*

Report of a Case

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THE treatment of hypertensive cardiovascular disease or congestive heart failure with a low-sodium diet is no longer considered an innocuous procedure. The recent observations of Soloff and Zatuchni[1] and of MacGuire[2] show that grave complications and death may ensue from sodium depletion.

The low-sodium syndrome is strikingly similar to acute adrenocortical deficiency. The clinical picture may develop with severe restriction of dietary salt, the use of mercurial diuretics that inhibit tubular reabsorption of sodium and intrinsic renal disease, such as tuberculosis of the kidneys or, rarely, chronic glomerulonephritis.[3]

Another complication of a low-sodium diet, not previously reported, is the flare-up of quiescent miliary tuberculosis. In the following case Addison's disease (chronic adrenocortical deficiency) and miliary tuberculosis were precipitated by a low-sodium diet for hypertensive cardiovascular disease.

Case Report

C. B., a 63-year-old housewife, was in good health during February, 1948, except for occipital headaches and subjective vertigo, for which she had been receiving treatment intermittently since 1944. During these years her blood pressure varied from 222/120 to 160/108. Medication had consisted of phenobarbital and the vasodilator group of drugs. In March a blood pressure of 220/110 was found. She was placed on a low-sodium (400-mg.) diet and reported complete relief of her symptoms several weeks later. Her blood pressure then was 140/90. From this point on she consulted numerous physicians, at varying intervals.

In April she believed that she was losing ground; she was weak and had lost considerable weight, the exact amount not being known. In June she became anorexic and there were days when she felt nauseated but she remained on the low-sodium diet. In August she noticed darkening of her skin. On August 15 she began to vomit and refuse food. She was admitted to the hospital on September 4.

The patient was acutely ill. Although conscious and apparently aware of her surroundings her lassitude[4] was so marked that she did not utter a sound. With no history accompanying her, the diagnosis was made on physical examination alone.

There was a bluish-bronze pigmentation of the face; the lips were cyanotic, and the respirations rapid. The pulse was not palpable, the heart sounds were inaudible, and the blood pressure was unobtainable. There was a distinct patch of bronze pigmentation on the buccal mucosa. The liver and spleen were not palpable. There was no peripheral edema.

Intravenous administration of saline solution was begun immediately, the presumptive diagnosis being an Addisonian crisis. Aqueous adrenocortical extract was given intravenously through the infusion tubing, and 5 mg. of desoxycorticosterone acetate was given intramuscularly. An hour later the patient had sufficient strength to speak in a whisper.

The same therapy was continued, the patient improving each day. By September 13 she was eating a large tray of food, taking salt tablets by mouth, and receiving 3 cc. of Lipo-Adrenal Cortex extract intramuscularly daily.

Fever was noted on her second hospital day, the temperature ranging from 99 to 104°F. Although tuberculosis was suspected, it could not be proved. Penicillin therapy was

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ineffective. A roentgenogram of the chest taken with a portable machine was normal. Anaerobic blood cultures were reported negative. No guinea pigs were available for inoculation. Acid-fast stains of the urinary sediment were negative. The eye grounds were inspected for miliary tubercles but none were found, the eye grounds being considered normal. The sedimentation rate was elevated, 32 mm. in 1 hour, the normal range being 15 mm. or less.

Examination of the chest on September 24 revealed dullness to percussion at the bases. Another roentgenogram on the next day confirmed the findings, the film being interpreted as "suggestive of miliary tuberculosis."

Administration of streptomycin hydrochloride, 250 mg. every 6 hours, was begun on September 25, and by September 28 the temperature range was not above 99°F., remaining so for the rest of her hospital stay.

The patient gained weight, and her color improved. It was difficult to persuade her to remain until a minimal course of streptomycin could be given.

On September 7 the specific gravity was 1.026, with negative tests for sugar and acetone and a + test for albumin, and the sediment contained heavy amorphous urates; 3 days later the specific gravity was 1.012, with negative tests for sugar and acetone and a + test for albumin, the sediment containing 5 to 10 white blood cells per high-power field. Stains for acid-fast bacilli were negative. (Guinea pigs were not available.) Blood cultures taken on September 21 and October 8 and 23 were reported negative after 3 weeks. On September 22 agglutination tests for typhoid, paratyphoid, *Proteus OX19* and *Brucella abortus* were negative in all dilutions. A glucose tolerance test showed a fasting blood sugar of 80 mg. per 100 cc. initially, 100 mg. after 1/2 hour, 106 mg. after 2 hours and 136 mg. after 3 hours. Pertinent laboratory data are presented in Table 1.

A roentgenogram of the chest on October 18 showed no change from the previous film. The sedimentation rate on October 23 was 22 mm. in 1 hour, a distinct improvement. The blood pressure was 136/80 and remained at that level during the rest of her stay.

The patient was discharged on October 26. The total dosage of streptomycin was 30 gm. given in 30 days. A relative of the patient was instructed to inject 1 cc. of Lipo-Adrenal Cortex extract daily.

The patient was readmitted to the women's medical ward on February 2, 1949. She had remained well for several months, but a combination of circumstances, both emotional and economic, had led to neglect of diet and irregular injections of adrenocortical extract. She became weak and complained of nausea. One week before admission she complained of pain in the right loin.

On admission the patient was disoriented, dyspneic and incontinent. A roentgenogram taken with a portable machine was reported as showing a heart enlarged in all diameters. The right leaf of the diaphragm was elevated, and an area of increased density at the right base suggested a localized pneumonitis. Some generalized coarse mottling of the lungs was noted.

Many examinations of the urine were negative except for a trace of albumin. The specific gravity ranged from 1.006 to 1.020. Blood chemical and hematologic values were similar to those observed on the first admission.

The patient gradually improved under the same therapeutic regime followed during her first admission. In addition, because of heart failure precipitated by the toxemia and pneumonitis, she was digitalized (February 5). Two small blood transfusions of 250 cc. each were given to combat the prostration and anemia. By February 7 she was able to take fluids and salt by mouth.

It was believed at this time that her maintenance requirement of desoxycorticosterone acetate should be estimated and, when she was strong enough, a pellet implanted.

Streptomycin was discontinued on February 14 because of complaints of dizziness. There had been no fever on this admission so that temporary interruption of the streptomycin course was considered fairly safe.

The patient's progress continued. She looked forward to returning home. On February 25 she vomited once. Despite the fact that laboratory studies were within normal range she appeared weaker. Adrenocortical extract was substituted for the desoxycorticosterone acetate on February 28.

The patient died suddenly on March 2.

At post-mortem examination* a pale, sallow, yellowish discoloration of the skin resembling that seen in Addison's disease was observed. There was no marked discoloration of the mucous-membrane surfaces except in focal areas. The extremities were otherwise not remarkable. A healed wound was present over the right internal malleolus — the result of a previous attempt at intravenous injections of fluid. There was external evidence of edema.

When the pleural cavity was opened the left hemithorax contained some dense fibrous adhesions, some of which could easily be broken. Within the left pleural cavity there was approximately 800 cc. of a yellow serous fluid. The right pleural cavity also contained many dense adhesions, which could be broken with difficulty, and 150 cc. of the same type of yellowish fluid. The lungs appeared normal in shape but were firmer in consistence than usual. The right lung weighed 270 gm., and the left 430 gm. On section the left lung exuded a bloody, frothy serous fluid. The pleural surface of the right lung was covered by dense fibrous tissue, which was somewhat edematous. Numerous small miliary

*Performed by Drs. William R. Platt and John F. Fitzpatrick.

TABLE 1. *Pertinent Data on the First Admission.**

DATE	RED-CELL COUNT	HEMOGLOBIN	WHITE-CELL COUNT	NEUTROPHILS	NON-SEGMENTED NEUTROPHILS	LYMPHOCYTES	MONOCYTES	EOSINOPHILS	BASOPHILS
	x10⁶	*gm./100 cc.*	*x10³*	%	%	%	%	%	%
9/5/48	4.96	15.6	6.3	66	3	30	1	—	—
9/7/48	—	—	—	—	—	—	—	—	—
9/10/48	—	—	—	—	—	—	—	—	—
9/12/48	3.17	12.9	7.4	52	2	41	1	4	—
9/15/48	—	—	—	—	—	—	—	—	—
9/19/48	3.52	12.8	7.6	55	1	41	1	—	2
9/21/48	—	—	—	—	—	—	—	—	—
9/22/48	—	—	—	—	—	—	—	—	—
9/25/48	3.35	11.4	6.2	43	—	55	—	2	—
10/1/48	—	—	—	—	—	—	—	—	—
10/6/48	4.27	10.5	—	—	—	—	—	—	—
10/7/48	—	—	—	—	—	—	—	—	—
10/8/48	—	—	—	—	—	—	—	—	—
10/13/48	4.02	12.8	5.4	57	—	36	2	5	—
10/19/48	4.20	12.8	6.2	49	—	42	3	6	—
10/23/48	—	—	—	—	—	—	—	—	—

*An electrocardiogram on September 9 showed an auricular rate of 95 and a ventricular rate of 95, with a PR interval of 0.18 second, QRS complex of 0.08 second, normal P wave, small Q wave in Lead 2 and pronounced Q wave in Lead 3, low-voltage R waves and normal ST segments and T waves (changes frequently seen in coronary sclerosis). On October 18 the cardiac rate was 90, with a PR interval of 0.20 second and QRS complex of 0.06 second and R waves of decreased amplitude (these changes, in association with left-axis deviation, were regarded as abnormal).

grayish-white areas measuring 4 to 6 mm. in diameter were seen throughout the cut surface of the right lung.

The heart weighed 410 gm. The pericardial cavity and the pericardium proper were not remarkable. Examination of the coronary vessels showed moderate sclerosis but no evidence of any occlusion. The auricular and ventricular cavities were not remarkable. Examination of the valves, endocardium and myocardium showed no significant pathologic changes.

The liver weighed 1730 gm. and had a mottled appearance. Beneath Glisson's capsule there were numerous small, pin-point miliary foci. On section they resembled the lesions seen in tuberculous infections. The intervening hepatic tissues appeared somewhat congested.

Examination of the gastrointestinal tract showed no significant pathologic changes except for marked edema of the subperitoneal tissues, especially those on the abdominal wall and within the mesentery proper.

Throughout the cortical surface of the spleen there were numerous grayish-white streaks, which on cut section appeared well defined. There was also marked hyperplasia of the malpighian bodies, with associated congestion of the sinuses. The spleen weighed 300 gm.

No significant gross pathologic changes were observed in the lymph nodes examined.

The perirenal tissues were somewhat edematous, but the kidneys were normal in position; the right kidney weighed 150 gm., and the left 200 gm. The capsule stripped with ease, leaving a mottled, pinkish-gray surface studded with numerous miliary tubercles. On cut section the corticomedullary junctions were quite distinct and yellowish red. The arcuate vessels were quite prominent; the ureters and bladder showed no significant pathologic changes.

Examination of the reproductive system revealed the uterus, tubes and ovaries to be somewhat atrophic, without any significant gross pathologic changes.

The periadrenal regions showed marked edema with marked invasion of the cortex and medulla by numerous grayish-white tubercles. The intervening tissue was pink, and a somewhat yellowish viscid fluid exuded from the cut surfaces.

No significant gross changes were observed in the pancreas.

Permission was not given to open the head.

Microscopical examination revealed moderate sclerosis of the coronary vessels. Otherwise the heart-muscle tissue was not remarkable. There was marked serous exudation into the pulmonary alveoli, with numerous miliary tubercles throughout. There was no infiltration of the blood-vessel walls or any evidence of a pneumonic process.

Many miliary tubercles were seen in numerous zones of the hepatic lobules. The corticomedullary areas showed marked thickening of the smaller arterioles, with narrowing of some of their lumens.

No active inflammatory process was present, although occasional tubercles were found throughout the kidney parenchyma proper.

The normal adrenal architecture had been completely distorted by a diffuse tuberculous involvement. There were occasional deposits of pink-staining hyaline material resembling amyloid. Throughout the rest of the adrenal gland there was a moderately diffuse lymphocytic infiltration.

The splenic pulp showed numerous tubercles of the miliary type.

No significant microscopical changes were observed in the pancreas.

The final pathological diagnoses were miliary tuberculosis affecting all organs except the pancreas, possible amyloid deposits in the adrenal glands, chronic passive congestion of the liver, lungs and spleen and bilateral, moderate hydrothorax. Death was due to a combination of adrenal insufficiency and toxemia associated with miliary tuberculosis.

TABLE 1 (*Concluded*).

DATE	BLOOD SUGAR	BLOOD UREA NITROGEN	CARBON DIOXIDE	CHLORIDE	TOTAL PROTEIN	SERUM ALBUMIN	SERUM GLOBULIN	SEDIMENTATION RATE
	mg./100 cc.	*mg./100 cc.*	*%*	*milliequiv./liter*	*gm./100 cc.*	*gm./100 cc.*	*gm./100 cc.*	*mm. in 1 hr.*
9/5/48	—	—	—	—	—	—	—	—
9/7/48	125	30	25	275	5.5	—	—	—
9/10/48	67	—	48	—	4.36	2.4	1.96	—
9/12/48	—	—	—	—	—	—	—	—
9/15/48	80	10	—	445	—	—	—	—
9/19/48	—	—	—	—	—	—	—	—
9/21/48	—	—	—	—	—	—	—	32
9/22/48	—	—	—	412	—	—	—	—
9/25/48	—	—	—	413	—	—	—	—
10/ 1/48	—	—	—	462	—	—	—	—
10/ 6/48	—	—	—	—	—	—	—	—
10/ 7/48	—	—	—	396	—	—	—	—
10/ 8/48	—	—	—	—	—	—	—	32
10/13/48	—	—	—	446	—	—	—	—
10/19/48	—	—	—	479	—	—	—	—
10/23/48	—	—	—	495	—	—	—	22

DISCUSSION

The necessity of an intact adrenal cortex for normal sodium metabolism and electrolytic balance is now well appreciated.[5] The converse, that a depletion of blood sodium disrupts the homeostasis of the adrenal cortex, may also be true. When hypoadrenocorticalism cannot be proved by formal studies, the provocative low-sodium test of Wilder has been utilized, extending over five days at most. A procedure of this sort extending over many weeks might disrupt adrenocortical function, and this in turn might lower the local defense mechanisms, activating a quiescent miliary tubercle.

The unusual clinical course of this patient can best be explained by the activation of miliary tubercles in the adrenal glands. Such latent tubercles are not uncommon. In routine autopsies they may be missed. Hardwig and Wright[6] reported 5217 consecutive autopsies in a general hospital. They observed miliary tubercle involvement of the adrenal glands in 78 (1.48 per cent), and yet only 3 of these patients had clinical Addison's disease. Reichle[7]

described latent tubercles in such structures as the spleen, liver, kidneys, lungs and lymph nodes in 20.1 per cent of 452 routine autopsies.

Proof that this patient did not have subclinical Addison's disease or active miliary tuberculosis before she undertook a low-sodium diet is afforded by the statements of her physicians, the existence of a moderately severe hypertension, and her own statement of relatively good health corroborated by her photograph taken a few weeks before she began the low-sodium diet.

The enlarged heart found at autopsy — at variance with the small heart usually found in Addison's disease — is explained by the previous hypertension, which, therefore, must have existed for some time.

Renal tuberculosis played no part in the sodium depletion. Urinalyses were entirely negative, except for a low-grade albuminuria, and microscopical study of the sediment for acid-fast bacilli was negative. It was the opinion of the pathologist that the renal tubercles were of very recent origin and probably had no effect on renal function.

Only the unwillingness of the patient to remain in the hospital on her first admission prevented a longer course of streptomycin treatment. Bunn[8] reported 15 cures in 100 cases of miliary tuberculosis or tuberculous meningitis, the length of treatment extending from eleven to eighteen weeks and the dose of streptomycin being approximately 1.8 gm. daily.

Summary

A case is reported in which a long-continued low-sodium diet for hypertensive vascular disease was apparently the exciting factor in the development of Addison's disease and miliary tuberculosis.

I am indebted to Drs. Mark Brown and B. T. Bailey Flack, of Haddonfield, New Jersey, who kindly supplied the prehospital history of the patient.

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ESOPHAGOSCOPY IN UPPER GASTROINTESTINAL BLEEDING*

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THE prompt diagnosis of the cause of bleeding in patients with hemorrhage from the upper gastrointestinal tract is difficult. Patients bleeding from esophageal varices must be distinguished from those bleeding from other lesions because prognosis as well as therapy depends on the cause of bleeding. Furthermore, a decision for emergency surgery requires early diagnosis. This paper illustrates the value of esophagoscopy in the diagnosis of gastrointestinal hemorrhage.

Method

During a ten-month period members of the Thoracic Surgery Service of the Boston City Hospital performed esophagoscopy on 14 patients with severe upper gastrointestinal bleeding. Nine of these 14 patients were examined endoscopically while in bed on the ward because they were too ill for transfer to the operating room. The detailed technic of emergency bedside endoscopy has been reported elsewhere.[1] A brief summary of the procedure follows:

All patients were given 50 to 100 mg. of Demerol subcutaneously half an hour prior to the examination. The lips, base of the tongue and oropharynx were anesthetized topically with 3 to 6 cc. of 2 per cent Pontocaine Hydrochloride. With minimum movement, the patient was placed in supine position with the head dorsiflexed, and the Jackson esophagoscope was gently passed. Esophageal varices, when present, were readily identified as blue, dilated, tortuous veins.

When bleeding varices were found, oxycel-gauze tampons were introduced through the esophagoscope for compression of the varices. The esophagus was packed with 6 to 12 tampons beginning at the bleeding point and continuing upward as the esophagoscope was withdrawn slowly. One patient with esophageal packs was given nothing by mouth for twenty-four hours

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after esophagoscopy. Oral intake was prohibited for two hours in those without bleeding varices.

Results

Table 1 summarizes the data obtained from 14 patients studied. These patients ranged in age from thirty-eight to eighty-eight years, with an average age of sixty-six years. There were 11 males and 3 females. Endoscopy excluded the diagnosis of esophageal varices in 7 patients with known or suspected cirrhosis of the liver (Cases 3, 4, 5, 11, 12, 13 and 14). Emergency gastric surgery was subsequently performed on 4 of these (Cases 3, 4, 12 and 13).

A presumptive diagnosis of bleeding esophageal varices was confirmed by esophagoscopy in 3 patients with proved cirrhosis (Cases 1, 7 and 8). Other bleeding lesions, for which surgery might have been indicated, were thereby excluded. Oxycel-gauze tamponade of actively bleeding varices was instituted in one of these, with apparent arrest of bleeding (Case 7).

Table 1. *Pertinent Data in 17 Patients.*

Case No.	Age	Sex	History	Post-esophagoscopy Diagnosis	Final Diagnosis	Discomfort	Place of Examination
1	68	M	Cirrhosis and bleeding esophageal varices proved 16 yr. before; hematemesis 3 times and melena 4 times 1 day prior to admission, continuing to death 4 days after admission.	Bleeding esophageal varices	Varices with bleeding point and cirrhosis of liver (autopsy)	Minimal	Ward
2	46	M	20-yr. history of duodenal ulcer with perforation 10 yr. previously; recurrent symptoms for 3 mo.; hematemesis, melena and fainting 2 hr. prior to admission; emergency gastrectomy 2 days after admission with recurrent melena and hematemesis 10 days postoperatively; dilated veins seen on stomach at operation raised question of esophageal varices as cause of postoperative bleeding.	No varices; blood seen in stomach.	Hemorrhage from pancreaticoduodenal vein in unresected duodenal ulcer (autopsy)	Minimal	Ward
3	70	M	Gnawing epigastric distress for 2 mo., melena 1 wk. and bright blood by rectum 1 day prior to admission; chronic alcoholism with 18 per cent bromsulfalein retention; emergency operation contemplated if varices excluded.	Normal esophagus	Duodenal ulcer (subtotal gastrectomy); carcinoma of sigmoid (sigmoidectomy).	Moderate (cervical arthritis)	Ward
4	44	M	Hematemesis and melena 2 days prior to admission and 1 day in hospital; chronic alcoholism with 23 per cent bromsulfalein retention; x-ray diagnosis of gastritis and duodenitis; gastric surgery contemplated; varices to be excluded.	Normal esophagus	Gastritis and duodenitis (sub-total gastrectomy); cirrhosis of liver (biopsy).	(General anesthesia because of incipient delirium tremens)	Operating room
5	82	M	5 yr. history of epigastric pain recurring 5 days prior to admission; hematemesis and melena day of admission; 3 gastrointestinal x-ray series equivocal for varices; 18 per cent bromsulfalein retention.	Normal esophagus	Duodenal diverticulum and (?) chronic duodenal ulcer on x-ray examination	Minimal	Operating room
6	88	M	4 yr. of pain from proved duodenal ulcer; severe pyrosis for 1 yr. increasing 5 wk. before admission; nausea 5 wk. and melena 5 days before admission; esophagitis suspected; hiatus hernia seen on x-ray film.	Severe esophagitis oozing blood on mucosal contact	Hiatus hernia; esophagitis and (?) duodenal ulcer on examination.	Moderate	Operating room
7	67	F	Massive hematemesis 4 times in 8 mo. requiring 21 transfusions for esophageal varices demonstrated in x-ray study; known cirrhosis with 18 per cent bromsulfalein retention; esophagoscopy with oxycel tamponade done as emergency measure.	Bleeding varices apparently controlled by oxycel	Esophageal varices (x-ray examination) and cirrhosis of liver	Minimal	Ward
8	58	F	Cirrhosis, ascites and jaundice in patient with severe chronic alcoholism with recurrent hematemesis and melena; esophageal varices suspected.	Varices seen	Esophageal varices (x-ray examination) and cirrhosis of liver	Moderate (incipient delirium tremens)	Ward
9	81	M	5 yr. of recurrent acute and chronic melena, pyrosis and upper abdominal pain relieved by standing upright. Hiatus hernia by x-ray examination; (?) esophageal ulcer.	Normal esophagus	Hiatus hernia (x-ray examination)	Minimal	Operating room
10	70	M	Hematemesis 6 yr. previously; hematemesis and melena 1 week prior to admission; x-ray films showed duodenal diverticula and (?) varices on previous admission.	Normal esophagus; blood oozing from stomach.	Chronic gastritis with erosions; duodenal diverticula (gastrectomy).	Minimal	Ward
11	38	M	Known alcoholic patient on spree for 3 wk. prior to admission; massive hematemesis on day of admission; 10-mo. history of postprandial distress relieved by sodium bicarbonate; hepatomegaly and shock on admission; (?) varices.	Normal esophagus; blood oozing from stomach.	(?) Gastric erosions; (?) duodenal ulcer (x-ray examination).	Minimal	Ward
12	65	F	3 mo. of epigastric pain; 3 days of melena and 1 day of hematemesis; liver normal in size; 17 per cent bromsulfalein retention; operation contemplated.	Normal esophagus	Bleeding gastric ulcer (gastrectomy)	Minimal	Ward
13	66	M	Suprapubic prostatectomy for benign prostatic hypertrophy with urinary retention; massive hematemesis and melena 6 days postoperatively; history of alcoholism; large, palpable liver; emergency gastric operation contemplated but delayed by clinical diagnosis of esophageal varices.	Normal esophagus	Bleeding duodenal ulcer (gastrectomy)	Minimal	Ward
14	67	M	Known cirrhosis (nonalcoholic) for 2 yr.; severe melena 5 days prior to admission; 21 per cent bromsulfalein retention.	Normal esophagus	Gastritis; duodenal ulcer (x-ray examination).	Minimal	Operating room

Esophagoscopy was performed on 2 patients with melena of unknown origin. These patients also had pyrosis and dysphagia, which suggested esophageal disease. Hiatus hernia was demonstrated by x-ray films in both patients. Esophagoscopy revealed severe esophagitis in 1 case and a normal esophagus in the other.

The presence of esophageal varices identified at endoscopy was proved by autopsy in 1 patient and reaffirmed by later x-ray examination in 2 patients. Extraesophageal bleeding sites were identified by x-ray examination, operation or autopsy in the 10 patients in whom a normal esophagus was found by endoscopy. In the remaining case of esophagitis there was no opportunity for confirmation.

Local anesthesia was used on 13 of the patients. One (Case 4) was given topical anesthesia, and the procedure was discontinued when it became apparent that the agitation of incipient delirium tremens contraindicated examination under topical anesthesia. Four patients were receiving blood transfusions and nasal oxygen while esophagoscopy was being performed.

Discussion

Prompt recognition of patients bleeding from esophageal varices is urgent. Diagnosis based on clinical grounds alone, however, is not always reliable, and some patients may be too ill for transfer to the x-ray examining room. In patients bleeding massively from peptic ulcer emergency surgery may be life saving. Surgery is contraindicated, however, in patients bleeding from esophageal varices. Esophagoscopy is a definitive diagnostic procedure in such cases.[2]

As an emergency measure esophagoscopy was performed, under topical anesthesia, on 9 patients in bed on the hospital wards. Delayed or interim esophagoscopy, as recommended by Jackson,[3] was reserved for 4 patients with less severe bleeding in whom the diagnosis of esophageal lesions such as varices, ulcer, tumor and inflammation was suspected but unconfirmed because of equivocal roentgenographic findings. Esophagoscopy was performed on these patients in the operating room under topical anesthesia. One patient who was considered for emergency gastric surgery was examined in the operating room under general anesthesia because he failed to co-operate under local anesthesia. When a normal esophagus was found, the general anesthesia was continued for the gastric resection.

Effective use of esophagoscopy requires close cooperation between attending physician and endoscopist. In this clinic, where many cases of upper gastrointestinal bleeding occur in chronic alcoholic patients, the possibility of ruptured varices arises frequently. The bromsulfalein-retention test has facilitated the early recognition of varices by screening patients with severe liver disease from those without.[4] Persistently elevated retention has been considered an indication for esophagoscopy in bleeding patients. Such patients are not operated on until bleeding varices have been excluded. In no case of the present series was the endoscopic observation found to be in error.

In spite of the advanced age and the severe illness of the patients examined, they complained of little discomfort. When present, discomfort consisted of slight distress caused by the extreme dorsiflexion of the head for esophagoscopy and occasionally a choking sensation as the esophagoscope displaced the larynx anteriorly when passing the cricopharyngeus muscle. Except for an occasional sore throat lasting twenty-four hours after the procedure, there were no complaints of pain. Cervical arthritis and advanced kyphosis may complicate esophagoscopy by producing increased discomfort, greater risk of perforation by pinching the esophagus between a bony spur and the instrument and limitation of examination of the entire esophagus. These factors limited the examination in only 1 patient (Case 6) of this series.

The danger of esophageal perforation and the need for meticulous endoscopic technic have been emphasized.[3, 5] In this series, complete topical anesthesia was always applied, an experienced head holder was always used, and the patient was never restrained. The operator reassured the patient throughout the procedure, and the examination was promptly discontinued if the patient failed to co-operate perfectly at any time. There were no deaths or complications from this procedure.*

Esophagoscopy did not precipitate recognizable bleeding in any patient with esophageal varices. Oxycel gauze was kept available for direct application should bleeding varices be found at the time of examination.[6] We have deliberately passed the esophagoscope for the therapeutic application of oxycel gauze in 1 patient only, known to be bleeding from previously diagnosed varices. There was apparently complete cessation of bleeding in this case. Intraesophageal balloon tamponade[7] was not used in this series.

Conclusion

Esophagoscopy contributes to the management of patients with massive gastrointestinal bleeding by facilitating the prompt diagnosis of esophageal varices in selected patients. Unwarranted gastric surgery may thereby be prevented. The early exclusion of varices in bleeding patients with known or suspected cirrhosis may expedite prompt operation when indicated.

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*There have been no significant complications up to this time after esophagoscopy for all indications performed by the Thoracic Surgery Service at the Boston City Hospital. Over 150 examinations, mostly on debilitated patients, have been performed annually for the past three years.

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ARCUS SENILIS AND SERUM CHOLESTEROL LEVELS IN THE ALEUT*

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BOSTON

ARCUS senilis is considered to be one of the stigmas of age not only in European whites but also in persons of all racial groups. In recent years investigators have become interested in determining what aspects of aging are responsible for arcus senilis, and they have reported a higher frequency of appearance in arteriosclerosis, hypertension, diabetes, xanthomatosis, and other disorders in which metabolic abnormalities superimpose premature aging.

Recently, Boas et al.[1-4] have devoted considerable attention to arcus senilis in relation to disorders of cholesterol metabolism, and they have demonstrated a higher incidence in persons with hypercholesterolemia, as well as noting that males were affected earlier and to a greater degree. They have assumed a causal relation between augmented serum cholesterol levels and arcus senilis, noting that this relation need not be invariable — that is, the correlation was not a perfect correlation.

The possible objections to their conclusions are not on the level of the facts and their interpretation, but on the design of the experiment.[5] They have, in effect, sorted on the basis of hypercholesterolemia by selecting patients with xanthomatosis, familial hypercholesterolemia, arteriosclerosis, coronary-artery disease and angina pectoris. It is possible that the relation between hypercholesterolemia and the arcus in such conditions is a secondary one operating through other metabolic disorders.

The ideal confirmatory test for this relation would be to select subjects on the basis of the arcus senilis alone, after having made certain that they are racially, economically, nutritionally and chronologically similar. Then, if the correlation existed in the absence of these diseases, one would find that the subjects with arcus senilis showed higher cholesterol levels than those without.

An opportunity to conduct such a sorting presented itself during the preparation of anthropologic and biochemical studies on 133 Aleut examined during the 1948 Peabody-Harvard Aleutian Expedition. These natives from Atka and Umnak Islands were racially homogenous; they showed panmixia on the basis of serologic tests[6]; and they lacked economic stratification and were on a common high-protein, low-vegetable diet. The present communication deals with the occurrence, age and sex distribution of arcus senilis in this group, and the cholesterol levels in those with and without the stigma.

Methods

During the field studies the eyes of all the Aleut studied in this series were examined with the aid of a small flashlight; details of pigmentation and patterning as well as the arcus were noted routinely on the Harvard anthropometric blank. In addition, Kodachrome eye photographs were made in many cases, and black-and-white photographs were made in all cases. Several degrees of arcus, as recorded by us, are shown in Figures 1 and 2.

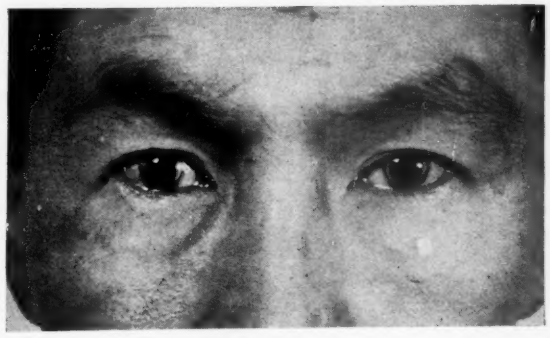

FIGURE 1. *Bilateral, Well Developed Arcus Senilis in a Forty-Two-Year-Old Aleut Man.*

Determinations of serum cholesterol were made, with the use of the Bloor technic, by the Chemical Laboratory of the Massachusetts General Hospital

*Research conducted in the Aleutian Islands under research sponsorship of the Peabody Museum of Harvard University, the Office of Naval Research and the Viking Fund. Data compiled at the Coronary Research Project, Massachusetts General Hospital, under a grant from the Commonwealth Fund of New York.

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on 81 blood samples collected by Dr. Fred Alexander, physician to the expedition. Control tests run by Miss G. Margaret Rourke, director of the laboratory, showed that no loss in cholesterol could be attributed to time or transportation from the Aleutian Islands.[7] Thus, cholesterol values reported here and in a separate communication on the Aleut[8] are comparable in laboratory technic to other values reported from this hospital.

FIGURE 2. *Partial Unilateral Arcus Senilis in a Fifty-Year-Old, Light-Eyed Aleut Man.*

RESULTS

Of 133 Aleut examined, ranging in age from eighteen days to seventy years, 17, or 13 per cent, exhibited various degrees of arcus senilis in one or both eyes (Table 1). All the 17 subjects showing arcus were over thirty years of age, with the exception of one eighteen-year-old youth from Atka. These findings are in agreement with still unpublished data from the island of Yap, collected by Dr. E. E. Hunt, Jr.[9]

TABLE 1. *Age Incidence of Arcus Senilis in 133 Aleut, of both Sexes, from Atka and Umnak.*

AGE GROUP	NO. OBSERVED	NO. WITH ARCUS SENILIS	PERCENTAGE WITH ARCUS SENILIS
yr.			
14*	55	0	0
15–19	12	1	8
20–29	23	0	0
30–39	15	2	13
40–49	14	5	36
50–59	7	5	71
60–70	7	4	56
All Aleut:			
0–70	133	17	13
15–70	78	17	22
30–70	43	16	38

*Range, 18 days to 14 yr.

The data collected in Table 1 show the clearly marked rise in the incidence of arcus senilis after the age of thirty, the majority of subjects over fifty years of age exhibiting this feature. Obviously, these figures are high compared to the incidence in whites in this country.

Table 2 demonstrates that among these Aleut arcus senilis is more than twice as frequent among males as among females, the numbers being 12 and 5 respectively. This is even more striking when it is noted that there are more females than males in the total group. Thus, the departure from the expected 1:1 ratio is quite significant. We have tested the departure from expectancy in this group by using the Chi square test. The Chi square value for 1 degree of freedom comes to 5.31, and this equals, on Fisher's tables, the 0.02 level of significance. Since we use the 0.05 level of significance, the disproportionate sex ratio in this study is judged to be definitely significant despite the small numbers involved. Arcus appears earlier and more frequently in the male.

TABLE 2. *Sex Ratios of all Aleut Examined and Those with Arcus Senilis.*

GROUP	NO. OBSERVED	NO. OF FEMALES	NO. OF MALES
All Aleut	133	77 (57%)	56 (42%)
Aleut with arcus senilis	17	5 (29%)*	12 (70%)†

*Mean age, 57.2 yr.
†Mean age, 45.4 yr.

This departure from chance distribution indicates that here, as in Boas's material, sex-influenced factors are involved in the appearance of arcus senilis; the frequencies preclude the possibility that they are sex-limited.

TABLE 3. *Cholesterol Values of Aleut over Thirty and of Eleven with Arcus Senilis.*

GROUP	NO.	AGE RANGE	MEAN AGE	MEAN CHOLESTEROL LEVEL ± STANDARD ERROR
		yr.	*yr.*	*mg./100 cc.*
Aleut over 30 yr. of age	30	30–70	46.6	204.2± 9.6
Aleut over 30 yr. with arcus senilis	11	30–70	53.6	199.1±15.7

Of the 81 cholesterol determinations made, 11 were on serums from persons exhibiting arcus senilis (Table 3). We have compared the mean cholesterol value for these 11 with the mean cholesterol value for 30 Aleut over thirty years of age; this was done in order to make the age of the two groups comparable since we have previously demonstrated an age-correlated rise in cholesterol in this group.[8] The difference between the two means, 5.1 mg. per 100 cc., is obviously not significant since it is smaller than the smaller standard error.

Thus, we may conclude that the Aleut with arcus senilis did not show elevated cholesterol levels.

It is interesting to note that there were 4 cases of hypercholesterolemia among the 81 determinations; only 1 of these subjects (a fifty-seven-year-old man) showed arcus senilis, and the probability was 7 out of 10 that he would have shown the stigma anyhow.

It is interesting to point out that arcus senilis frequently does not show comparable development in both eyes and may appear first in either eye. Thus, in the present group, 2 of the 17 showed unilateral development, the arcus senilis being in the left eye in 1 case and in the right in the other. In both cases the arcus senilis was rated as "slight" to "small," thus indicating a near-beginning stage.

Discussion

The experimental method of Boas et al. in demonstrating high frequencies of arcus senilis in hypercholesterolemia was to select first for degenerative disorders and disturbances of cholesterol metabolism. Having done so, they were able to conclude that "arcus senilis and xanthelasma in patients with coronary artery disease . . . are related to a disturbed cholesterol metabolism." The implication is, then, that the disturbance of the cholesterol metabolism must precede the appearance of arcus senilis, and that a group of subjects selected on the basis of arcus senilis alone should show an elevated serum cholesterol level *compared to others in the same group.* In the Aleut, at any rate, despite a high frequency of appearance of arcus senilis, hypercholesterolemia did not characterize the group with the stigma or the group as a whole. Unless it is assumed that the etiology of arcus senilis in Aleut, in the natives of Yap and other groups differs from that of arcus senilis in American whites, it must be concluded that comparative hypercholesterolemia is not a necessary precursor, and that arteriosclerosis and other degenerative diseases do not have to be present before arcus senilis develops.

Summary

In 133 native Aleut of Atka and Umnak Island arcus senilis was found in 17, or 13 per cent; 16 of these were over thirty years of age. The usual increase in frequency with age was noted. Arcus senilis was much more frequent and began earlier in males.

The mean cholesterol level for 11 Aleut with arcus senilis when compared to the mean level for all Aleut over twenty and all Aleut over thirty showed no evidence of elevation. Seven of the 11 showed serum cholesterol levels below 180 mg. per 100 cc. On the basis of the cholesterol studies and physical examinations, it was concluded that hypercholesterolemia or degenerative diseases, or both, need not be a concomitant of arcus senilis.

We are indebted to the United States Navy and the United States Coast Guard for assistance in obtaining the data, and to the advisory board of the Coronary Research Project (Drs. P. D. White, E. A. Hooton, E. F. Bland, J. Lerman, S. A. Levine and H. B. Sprague) for their advice and counsel. Mrs. Jennifer Lehmann made the statistical computations reported herein.

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MEDICAL PROGRESS

ECZEMA IN INFANCY AND CHILDHOOD*

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BOSTON

THE following discussion of the literature of the past four years concerns several aspects of eczema in infancy and childhood: pathogenesis, general considerations, antihistaminic drugs, milk-free diets, unsaturated fatty acids, treatment of secondary infection, bacitracin, fungi, scabies, Kaposi's varicelliform eruption and eczema vaccinatum, nummular eczema, seasonal eczema due to pollen, blood protein depletion and hospital morbidity of eczematous infants.

PATHOGENESIS

Atopic dermatitis comprises at least 75 per cent of the "eczema" of infancy and childhood, and in spite of much study the mechanism by which it is produced is still obscure. Although most patients with this disorder give multiple positive cutaneous test reactions of the urticarial type, the results of removal from the diet or environment of the allergens responsible for the positive tests are often so negligible that it is by no means clear in what way this urticarial type of sensitivity is involved in the production of the disease.

Rostenberg[1] finds that patients with atopic dermatitis react less frequently to simple chemical substances than those with eczematous dermatitis of the contact type do. It is not true that they do not give any type of patch-test reaction, for there is a relatively high incidence of positive patch tests to protein substances and to certain metallic salts. There is, in atopic dermatitis, an epidermodermal type of sensitivity other than the wheal type. This does not, however, indicate that the atopic and the eczematous, contact type of dermatitis are immunologically identical, and it may be true that the positive patch tests in these two conditions signify different types of epidermodermal sensitivity.

Rostenberg suggests that patients with atopic dermatitis have an immediate wheal type of sensitivity. Consequently, when they encounter the allergen to which they give the wheals they will have an antigen-antibody reaction, with the following effects of such a reaction: alteration of capillary permeability and release of histamine and probably other substances. There is also probably an epidermodermal hypersensitivity in these persons either to the same or to different allergens, but these allergens cannot get to the epidermis or cannot make their effects manifest until the capillary "door" is opened to them by the alterations in capillary permeability that result from the wheal type of reaction.

Rostenberg's hypothesis, in essence, is that in atopic dermatitis there is a dual hypersensitivity — dermal and epidermal. This seems reasonable, for most allergic people with hay fever or asthma have the same cutaneous wheal-test reactions that occur in atopic dermatitis, and still they only occasionally have dermatitis. It is plain, therefore, that something in addition to the wheal reaction is necessary for the development of dermatitis. It is by no means clear, however, just what this "something" is.

Hill,[2] in a critical review on the pathogenesis of infantile eczema, states that the disorder has elements of both atopic and contact dermatitis. Therefore, patch tests applied to the surface of the skin may sometimes supply useful information concerning the etiology. So may the urticarial type of reaction produced by scratch or intracutaneous tests, and they should not be discarded, as some have advocated. It is believed that there is ample proof that allergens giving this type of test produce eczema as well as urticaria.

Simon[3] has continued his interesting and valuable work with human dander. He finds that the incidence of positive patch, scratch or intracutaneous tests with human dander is much greater in patients with atopic dermatitis than in the general population or in patients with hay fever or asthma. In 1945 he[4] was convinced that exposure to human dander was the most important cause of infantile eczema. In 1947 he said[3]: "I have since studied a large series of cases quite carefully and find that diminished contact with human dander, as carried out clinically, has very little influence on the clinical course of the disease in the average case."

I do not agree with Simon's 1945 statement but do agree with his 1947 statement. Simon's work with human dander — and he has done a great deal of it and has done it very carefully — at present offers nothing that is of practical value in the management of infantile eczema. It is my opinion, however, that from a theoretical viewpoint this work has been very important, and that in the course of time, as it is further developed, it may well afford

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information that will clear up some of the obscure points in the pathogenesis of atopic dermatitis.

General Considerations

Six articles, written by a dermatologist and six pediatricians, all of whom have long been interested in this particular subject and whose collective experience with it has been enormous, are reviewed, not because any one of these men has made any startling progress or because anything really new is presented, but because it is valuable to get the point of view of those who have given a great deal of thought to the subject over many years.

Perlman,[5] in one of the best and most practical papers that has appeared in a long time, discusses infantile eczema from the point of view of a dermatologist. With the exception of the few cases in which food and inhalant allergens play important roles and in which their elimination solves the problem, the management of infantile eczema is little different from what it formerly was, and in spite of therapeutic advance, no specific remedy has been discovered. All eczemas begin as erythema, and after the initial stage of erythema (vascular engorgement and dilatation) edema, vesiculation, exudation and crusting occur. Various stages of the eczematous reaction are often present together. The pathologic process in eczema is reversible, so that the edema may be made to subside, with restoration of the normal skin. Perlman is a staunch advocate of careful local treatment, and thinks very little of the allergic approach: "Although removal of the offending allergen remains the ideal method of achieving the therapeutic result, experience has convinced most pediatricians that for the vast majority of patients that method of approach is a waste of time, money and effort." This is the opinion of many dermatologists. The pediatricians and allergists do not entirely agree with it, and although they realize that the immunologic approach is by no means ideal, and is often devoid of any practical results, it helps often enough so that it is decidedly worth while, and that if any progress is ever to be made in infantile eczema it will be made along these lines. The pediatricians and allergists, however, often do not pay enough attention to local treatment: here the dermatologists can teach them much. One of the reasons why poor results are too often obtained is that remedial agents are too often prescribed without a clear-cut indication for their use, and without regard to the stage of the eczema.

There is no need for the use of a large number of local medicaments — better results are obtained by the proper use of a few. All medicated "baby oils" should be avoided. Restriction of scratching is of the utmost importance and is best obtained by the use of arm splints.

During the acute stage wet dressings of boric acid or aluminum acetate solution, or one fourth of 1 per cent silver nitrate solution should be used, or if infection is present, a 1:10,000 or 1:20,000 solution of potassium permanganate. (Boric acid is best avoided; there is increasing evidence that if used over large surfaces it may be toxic, and when there are several other preparations that are as good, why should it be used?)

Better than any of these wet dressings, according to Perlman,[5] is the old-fashioned remedy, camomile tea. One quart of boiling water is poured over 4 heaping teaspoonfuls of camomile flowers — this is allowed to steep for twenty minutes and then strained, cooled and applied, as a wet-gauze dressing. In the subacute stage pastes containing crude coal tar or ichthamnol are advised, and in the third or chronic stage, in which there is much thickening, strong coal-tar or wood-tar ointments (3 to 6 per cent) are used. Vioform is also of considerable value: it is used in 3 per cent concentration in vaseline or zinc oxide ointment.

Ratner[6] emphasizes the fact that one of the first things to be done is to make a correct diagnosis, and to bear in mind that infants and young children have other skin diseases besides eczema, such as scabies, pityriasis rosea and drug eruptions. This is important. There has been a great deal of scabies in infants and young children since the war, and it is often incorrectly diagnosed, and treated as eczema.

Eczema is the most common allergic condition in infancy. Positive skin tests were obtained in 85 per cent of all patients under one year of age. Only 41 per cent reacted to foods alone — 58 per cent to a combination of foods and environmental allergens. Ratner believes that contact and atopic dermatitis have identical immunologic mechanisms and differ only in the mode of sensitization, so-called atopic eczema resulting from intrinsic contact with antigens, and contact dermatitis from external contact.

All patients are immediately placed on allergenically denatured diets fitting their needs. In these foods the albumin and globulin fractions have been coagulated and allergenically denatured by moist heat. The foods used are evaporated milk, Pablum, Pabena, thoroughly cooked vegetables, thoroughly boiled meats, hard-boiled egg and stewed fruit. Raw fruits, fish, nuts, condiments and chocolate are eliminated. Ratner has clearly shown by anaphylactic experiments in animals that foods treated in this way are less allergenic than raw foods. It must not be thought, however, that they are entirely nonallergenic: a strongly egg-sensitive infant is likely to react violently to hard-boiled egg white, nor is Pabena (oat), Pablum (wheat) or evaporated milk entirely nonallergenic.

In addition to omission of certain foods, careful attention to the environment is of importance. All dust-producing articles, including stuffed toys, should be eliminated, and pillows and mattresses

covered with impervious material. For secondarily infected, wet, oozing eczema, Ratner recommends the application of a 2 per cent aqueous solution of gentian violet. Wet dressings, as advocated by most dermatologists, are not of as much value in infants as in adults, and may lead to harmful chilling of the body. It is rarely advisable to use any proprietary ointment: the *United States Dispensatory* contains all the preparations that are necessary. Ammoniated-mercury ointment is recommended for infection. I agree with this, having used ammoniated-mercury ointment for thirty years without observing as much sensitization from it as has been reported by many observers from the use of sulfathiazole or penicillin ointment. It is hazardous to use sulfonamides, penicillin, streptomycin and tyrothricin ointments, for sensitization may occur, and innumerable cases have been reported. However, in severe infection these preparations should be used as they are in any severe infection — they should be given by mouth or hypodermically. The antihistaminic drugs may help itching somewhat, but they will not control the itching in a severe case. In no sense do they correct the eczema.

Glaser[7] stresses the fact that the treatment of infantile eczema is not entirely satisfactory. The problem is to do as well as possible with a disease that tends to eventual recovery regardless of therapy. It is one phase of a cycle that may go on with various manifestations (such as hay fever and asthma) for the rest of the patient's life.

Glaser recommends at the outset a simple elimination diet, the number of foods in it depending on the age of the child: a cow's milk substitute; one cereal, such as oatmeal; carrots and string beans; apple and pear; one meat (capon or rooster); and vitamins A, D and C.

Capon or rooster is specified to avoid feeding of any egg protein. If after about a week the child is better on this diet, one new food is added every few days, and the results are noted. Skin tests help in enabling one to select a diet that has the best chance of doing some good. No intracutaneous test should be done unless it has been preceded by a negative scratch test. This is important — it is a rule that should not be broken.

For itching, the antihistaminic drugs are only occasionally of benefit. For sedation, Glaser highly recommends Demerol. An infant over two months of age may be given one fourth of a 50-mg. tablet every four hours, and this dosage may be gradually increased if necessary. Children with eczema should be inoculated with diphtheria and tetanus toxoids in the same way and at the same time as any other child — they are no more likely than a normal child to have severe reactions.

Hill[8] likewise is of the opinion that the treatment of infantile eczema is by no means entirely satisfactory, partly because one is dealing with a patient who is working against him twenty-four hours a day (no skin can heal when it is continually rubbed and scratched) and partly because atopic dermatitis, which comprises about 75 per cent of what is called "eczema" in the young, has as its basis an abnormality of constitution that cannot be changed. Skin tests are of some help, but many positive tests, particularly to foods, are of no etiologic importance. Young children are often tested with as many as 200 foods, a large number of which they have never eaten, and never would eat. This is absurd. Cow's milk, especially in infants, is often important as a cause of eczema. The substitution of goat's milk rarely does any good, because there are so many crossed reactions between the proteins of these two milks. A milk-free food such as Mull Soy, Sobee or Nutramigen is likely to do better. These foods have a real place in the treatment of infantile eczema, but are too often prescribed for infants with an eczema that has nothing to do with milk. By no means all infantile eczema is caused by food: infants have contact dermatitis just as adults do, and it is not uncommon. Hill has seen it from the sizing in a new unlaundered undershirt, from the dye in a pink sweater, from medicated baby oil, from Aristol powder (thymol iodide), from hypochlorite bleaching powder, from the rubber waistband of a patent diaper and from furniture polish.

Ross and Brown[9] recommend boiled evaporated-milk mixtures as the most satisfactory type of feeding for infants with eczema due to milk sensitivity. (Evaporated milk, during processing, has been already heated to 240°F. for twenty minutes; it is not likely that it is rendered any less allergenic by further boiling, although this is often recommended.) In the experience of Ross and Brown substitutes for milk have not been very satisfactory, because they have not the nutritional value of milk, and weight gain and general progress are not likely to be good. For local treatment they highly recommend the following salve devised at the Hospital for Sick Children, in Toronto:

Substitute	Amount
	%
Crude coal tar	2.5
Titanium dioxide	7.5
Metaphen	1.0
Butesin	1.0
Zinc oxide	7.5
Hydrosorb base (as needed)	

Titanium dioxide is used for its strong dehydrating power, Metaphen for its antiseptic action, and Butesin for its antipruritic effects.

Bowen and Bloom[10] believe that skin testing is of little value in infants under one year of age. Indirect testing may be rarely of value, but in the main this procedure is only of "museum importance." Many cases will persist in spite of all dietary manipulation. Milk, wheat and egg are considered offenders until proved otherwise. In milk-

sensitive infants evaporated milk boiled four hours is first tried. Goat's milk is rarely of value. The authors have never found a severely milk-sensitive patient who could take evaporated milk in any form. In such cases it is necessary to use a milk-free diet. They do not believe that Pablum is nonallergenic. They very wisely say, "Remember you are treating a growing baby and not a skin test report."

Unsaturated fatty acid therapy does not give striking results, but is worth a trial for a two-week period. Antihistaminic drugs help somewhat in controlling mild itching.

For the acute oozing stage Bowen and Bloom use a 1:20 solution of aluminum acetate or a 1:500 solution of silver nitrate. When the oozing has been controlled they change to a crude coal-tar paste: this should not be used over more than 50 per cent of the body surface. For infection they use potassium permanganate baths, 1:10,000, and sulfadiazine by mouth or penicillin by injection, followed by a 3 per cent Vioform ointment. They advise the avoidance of mercurial antiseptics because of the danger of sensitization.

Antihistaminic Drugs

Every large drug house now has its own antihistaminic, each varying slightly in chemical composition, and each recommended by its producers as the "best." These drugs in the last four years have been taken by untold thousands of people for every conceivable allergic condition, and the literature is full of reports in the usual manner stating the percentage of patients "entirely, partially or slightly relieved." Such reports can never be accurate, but by a composite study of them it is possible to obtain a fair idea of the situation. It is generally agreed that these drugs are of very frequent value in hay fever and urticaria, and only rarely so in asthma. The reports on their value in atopic dermatitis are conflicting.

Baer and Sulzberger[11] treated with Pyribenzamine 56 patients who suffered from various itching dermatoses. Included among these were 11 cases of atopic dermatitis. Of these, 1 patient had good relief with Pyribenzamine alone, 5 had fair to excellent relief with Pyribenzamine in addition to other therapy, and 5 had no relief. The authors conclude that Pyribenzamine could not be shown to be of very great value in controlling the itching or the eruption of atopic dermatitis.

Lockey[12] treated 7 cases of atopic dermatitis in young children with Benadryl. There was slight improvement of the itching in 2 cases.

Levin[13] treated 60 cases of atopic dermatitis with Benadryl; there was moderate relief of itching in 6.

Waldbott[14] treated 6 cases of atopic dermatitis with Benadryl; there was relief of itching in 2.

Criep and Aaron[15] treated 21 cases of atopic dermatitis with Neo-Hetramine, which was of no value in 5 per cent; 40 per cent of patients had less itching, and 10 per cent were entirely relieved. In 12 children 10 per cent Neo-Hetramine ointment was used, with negligible results. The authors came to the following conclusion: "It is difficult to interpret these results because other treatment was used concomitantly, and because it was difficult to evaluate psychogenic factors."

Kesten[16] treated with Pyribenzamine 20 infants and children who had atopic dermatitis. There was considerable relief of itching in 15. Almost all slept for six or seven hours uninterrupted, at night, while taking the Pyribenzamine. However, the eczema did not clear until the sensitizing allergens had been removed from the diet and environment.

Levin and Moss[17] treated 4 cases of infantile eczema with Thephorin. In 3 cases there was about 75 per cent relief of the itching.

Wooldridge and Joseph[18] treated 23 patients with atopic dermatitis, varying in age from five months to fifty-two years, with Thephorin, both by internal administration and in the form of an ointment, with the following results: complete clearing, 2 cases; more than 75 per cent clearing, 7 cases; more than 50 per cent clearing, 8 cases; patient helped, but not much, 2 cases; no improvement, 3 cases; and condition made worse, 1 case.

Feinberg and Bernstein[19] were the first to report on the use of antihistaminics in ointment form. They treated 33 cases of atopic dermatitis, 10 in patients under ten years of age, with a 2 per cent Pyribenzamine ointment. There was relief of itching in 24 cases. The authors found that relief was greatest when the eczematous lesions were localized in small areas.

For 22 patients with various itching dermatoses, Perry[20] employed a 2 per cent Benadryl ointment. Of these patients 8 had atopic dermatitis. Moderate relief of itching was obtained in 6, and excellent relief in 2. Of the 8 patients, however, 4 obtained the same relief with the ointment base alone.

Epstein and Macaulay[21] used a 2 per cent histadyl cream in 6 cases of infantile eczema. It was of help in 3 cases, and did no good in 3. The best indication for the antihistaminic ointments was considered to be a localized eruption, such as circumscribed neurodermatitis.

Sulzberger, Baer and Levin[22] treated 16 cases of localized atopic dermatitis with Pyribenzamine ointment. In 8 of these there was marked relief. Of 40 disseminated cases only 2 showed slight improvement. The control of itching is one of the most important and difficult problems in dealing with infantile eczema, and in my opinion there is at present no really good answer to it. If the antihistaminic drugs taken internally worked consistently in a large proportion of cases, they would be ideal. In my experience, they do not do much good unless they happen to make the baby sleepy, which does not occur very often. The itching in

most cases of infantile eczema is not localized; it is scattered all over the body, so that local applications are not likely to do any good unless the baby is kept at all times covered from head to foot with the ointment. Furthermore, the antihistaminic ointments do no good to the eczema itself.

From this brief review of only a few of the typical papers that have been written on the subject, it is apparent that an antihistaminic drug given internally may sometimes help itching, and is certainly worth a trial, but too much must not be expected of it. The use of these drugs in ointment form is also worth a trial if the dermatitis is localized — not much benefit is likely to accrue if it is widely scattered.

Milk-Free Diets

Wolpe[23] fed 74 eczematous infants with various milk substitutes — nine different preparations were used. He concludes that those fed on milk substitutes reached a clinical relief faster than those fed on milk.

Stoesser[24] fed 37 patients on a soy-bean preparation. Of the 37, 29 took it well. There was looseness of the stools in 14. Of the 29 who took it well, the eczema was greatly relieved or cured in 18. Only 5 patients gave positive cutaneous tests to milk protein. (If the intracutaneous test had been used there would have been more positive reactions.)

Unsaturated Fatty Acids

In 1929 Burr and Burr[25] reported a new deficiency disease produced in rats by a fat-free diet. This was manifested by a dry, scaly dermatitis, and could be prevented or cured by the addition of unsaturated fatty acids to the diet, contained in corn oil, linseed oil or lard. In 1933 Hansen[26] applied these findings to infantile eczema, and found that in eczematous infants the unsaturated fatty acid content of the blood was low and could be raised by the ingestion of corn oil, linseed oil or lard, and that coincident with this there was improvement in the skin condition.

Since that time a number of investigators have used this treatment in atopic dermatitis, mostly of adults. Some have confirmed Hansen's findings and some have not, and unsaturated fatty acid therapy has not been adopted as a standard treatment for atopic dermatitis.

Except for the original reports of Hansen and those who have worked with him, there has been little or nothing in the literature concerning this mode of therapy for the last few years, until 1947, when Hansen and his collaborators[27] reported a longer and very carefully studied series of cases. In the series there were 148 infants and children with eczematoid eruptions — no attempt was made to classify them. Usually at the outset one teaspoonful of lard was given three times a day — this was gradually increased to 2 or 3 teaspoonfuls three times a day for the larger children. Similar amounts were given when corn oil or linseed oil was used. This was continued for two or three months before therapeutic evaluation was attempted — results do not come quickly.

Of the 148 patients results were good to excellent in 60, fair to good in 51 and poor in 37. Young infants responded best. Coincident with improvement of the skin there was usually increase in the iodine number of the blood serum fatty acids. This increase was also noted in patients who were being given local treatment with crude coal tar, but no lard, linseed oil or corn oil. It does not seem likely to the authors that eczema is the direct result of a diet deficient in fat, but that the essential fatty acids are required for maintenance of the normal integrity of the skin, and that other factors may be influential in the development of the dermatitis. They say that this method of treatment is merely an adjunct, and should be combined with all the usual measures employed in the treatment of eczema.

(To be concluded)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

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CASE 36081

PRESENTATION OF CASE

A forty-four-year-old married woman entered the hospital for the first time because of sudden loss of feeling in her legs.

Three days before admission she developed a gastrointestinal upset, with some nausea and diarrhea. These symptoms persisted until the time of admission. Just prior to entry she suffered an attack of vomiting following which she collapsed on the bed. Numbness and tingling suddenly occurred in the feet and crept up the legs so that the patient was unable to feel her legs at all. There was no severe pain with this but only a sensation that her feet were "sticking up in the air." She was able to move her feet slightly.

She had a known history of rheumatic heart disease and auricular fibrillation for fifteen years but had been busy and active without symptoms of diminished cardiac reserve other than exertional dyspnea on climbing two flights of stairs. The past history was otherwise not remarkable.

Physical examination showed a well developed, well nourished, acutely distressed woman who was able to move the legs only slightly. The skin except for the legs was warm and moist. The head and neck were not remarkable. The neck veins were flat. The lungs were clear. There was no dyspnea or cyanosis. The heart was enlarged to the left anterior axillary line and was fibrillating at a rate of 110. The mitral first sound was loud; the pulmonic second sound was reduplicated. There was a Grade IV apical diastolic murmur without presystolic accentuation. The abdomen was soft and nontender. The liver was not palpable. The legs were waxy and white, with no palpable pulses in the femoral, popliteal or dorsalis-pedis regions.

The temperature was 98°F., the respirations 20, and the pulse 90. The blood pressure was 190 systolic, 90 diastolic.

Five hours after the sudden episode in the legs exploration through a left paramedian incision was performed, and emboli were removed from the bifurcation of the aorta, the right common iliac artery at its bifurcation and the left common iliac artery. Through separate incisions the common femoral arteries were explored, and the left one opened to remove a clot lying just above the bifurcation. Prior to operation 100 cc. of urine was removed from the bladder by catheter. Following operation there was good and complete return of the pulse throughout the right leg, and the skin was warm and dry. The femoral and popliteal pulses were good in the left leg, but the posterior-tibial and dorsalis-pedis pulsations were not palpable. The patient was given whole blood transfusions and 800 cc. of 5 per cent dextrose in water with 10 mg. of heparin added.

Nine hours after operation the blood pressure had dropped to 90 systolic, 40 diastolic. The right leg was warm and dry. The left was cool three fourths of the way up the tibia. She was able to move the toes of the right foot but not those of the left. One hour later she was in collapse, the pulse and blood pressure being almost unobtainable. The lungs were clear, and the neck veins were flat. Laboratory studies at that time showed a hemoglobin of 15 gm. per 100 cc. The white-cell count was 34,600. The clotting time was 35 minutes in the first tube, 2 hours and 5 minutes in the second and 2 hours and 35 minutes in the third. The prothrombin time was 22 seconds (control, 18 seconds). The carbon dioxide was 19.2 milliequiv., the sodium 135.7 milliequiv., the chloride 98 millequiv. and the potassium 6.3 milliequiv. per liter. The nonprotein nitrogen was 57 mg. per 100 cc. Throughout the day despite three 500-cc. blood transfusions the blood pressure remained at levels of 60 to 70 systolic, 40 to 50 diastolic. There was no urinary output. There was no evidence of congestive heart failure. To combat the prolonged clotting time protamine sulfate and Hykinone were given.

During the course of the next twenty hours her condition remained essentially the same although the clotting time was shortened to 24 minutes in the first tube and 44 and 50 minutes in the second and third tubes. Gradually, with transfusions, the blood pressure rose during the second postoperative day, and the patient seemed improved but there was no output of urine. However, there were dullness and tenderness on the whole left side of the abdomen. There was mottling of the left foot, but the right appeared in good condition.

On the morning of the third postoperative day she seemed in better condition. There was then for the first time some distention of the neck veins, but the lungs were clear and the heart was essentially as before. A spinal anesthetic was given in an attempt to denervate the kidneys, and under

the same anesthetic an incision was made into the left flank and 300 cc. of blood was evacuated from a retroperitoneal hematoma. The clotting time was then 8.5 minutes. The nonprotein nitrogen was 108 mg. per 100 cc., and the chloride 78 milliequiv. and the potassium 8.8 milliequiv. per liter. By evening she was disoriented, the blood pressure began to fall again, and the respirations became irregular. Later she developed twitchings of the face, began frothing orally and died late that evening.

Differential Diagnosis

Dr. Conger Williams*: At first glance this looks too easy. I am sure there is some joker.

Let us begin with certainties. We are fairly certain that this patient had mitral stenosis and auricular fibrillation; therefore, it seems logical to connect this with the event that followed later and apparently brought on the acute illness — namely, emboli to the lower part of the main arterial trunk. This is a very common complication in mitral stenosis with auricular fibrillation. It is possible, however, that these clots came from elsewhere. Among other things to be considered is a mycotic aneurysm in the aorta higher up with emboli from that site. But, in view of the lack of clinical symptoms prior to the onset of the present illness, that seems extremely unlikely.

Other possibilities that we should consider include perhaps a silent coronary thrombosis with mural thrombus and emboli to the legs. She did have a so-called gastrointestinal upset before the really acute illness brought her into the hospital and very often myocardial infarction is mistaken for indigestion or other gastrointestinal disturbance. However, the fact that she had diarrhea seems somewhat against that. I think it is logical to say that the emboli — I judge they were emboli and not thrombi in situ — came from auricular thrombi associated with mitral stenosis. I might ask Dr. Linton whether or not it was quite clear that these were emboli.

Dr. Robert R. Linton: I think there is no question that these were emboli.

Dr. Williams: The patient was operated on, and clots were removed. Apparently, there were no untoward complications during the operation itself, but nine hours later the blood pressure dropped to just half its initial value and an hour after that it was almost unobtainable. That is something we must explain. Knowing that blood was evacuated from the retroperitoneal space later, one might wonder if this abrupt fall in blood pressure resulted from early bleeding. I think not, because a hemoglobin taken apparently at that time was reported to be 15 gm. per 100 cc. Even allowing for hemoconcentration as a result of shock, the hemoglobin level was high enough to make it unlikely that this drop in blood pressure was due to the hemorrhage. I know she had a hemorrhage, but I think that probably accounted for the later drop in blood pressure.

Another thing we must consider is the possibility of whether the retroperitoneal hemorrhage came from something other than the operative site. Knowing that she had vascular surgery, and knowing the difficulty of that procedure, I suppose that retroperitoneal hemorrhage came from the operative site, perhaps aided by the fact that the clotting and prothrombin times were prolonged. We sometimes see retroperitoneal hemorrhage from the rupture of an arteriosclerotic aneurysm. This woman seems rather young for that, even though she did have hypertension at the time of admission. A dissecting aneurysm might also be considered, but it would be very much of a coincidence to have a dissection accompany the embolic episode that was proved.

Other possibilities of hemorrhage include hemorrhage into the gastrointestinal tract. Again, I think this is unlikely at this time because of the finding of a hemoglobin of 15 gm. per 100 cc. It might explain a later drop in blood pressure but I doubt that it explained this one.

Another important point to consider is myocardial infarction with or without coronary thrombosis. Conditions were fairly good for such a complication. She had a very low blood pressure — 90 systolic, 40 diastolic — for nine hours, and then the blood pressure became almost unobtainable. I wonder if the first drop in blood pressure was produced by the operation, with blood loss of some extent followed by a second drop that was perhaps due to myocardial infarction. It is well known that the fall in arterial pressure that may follow a surgical procedure predisposes to myocardial infarction. No electrocardiogram is referred to in the record. I wonder if one was taken.

Dr. Tracy B. Mallory: None was taken.

Dr. Williams: Of course such a record would be very helpful in deciding whether or not she had myocardial infarction. Certainly, that diagnosis should always be considered in patients who develop a fall in blood pressure during or following operation.

Now there are other things I should like to ask about — why was the patient anuric for this length of time? Probably the very low level of the blood pressure accounted for this, but a favorite surprise finding in these sessions is that of emboli to the kidneys. No examination of the urine is reported in the record, but it has been shown repeatedly that the urine may be relatively normal in the presence of a very large renal infarction. I wonder if the source that produced the emboli responsible for the arterial occlusion also produced smaller emboli to the kidneys. It certainly is possible. However, with this low level of blood pressure for so many hours, I think the anuria may well be explained.

*Associate physician, Massachusetts General Hospital.

When I first read this protocol and saw the description of the early weakness of the legs, I wondered if we were going to have a case of potassium poisoning. However, developments in the record soon clarified that point; at least the weakness and numbness in the legs were not due to potassium poisoning. I think that the potassium level in the serum just before death could be explained by anuria. It is quite possible that the high potassium contributed to death, but certainly it was not the main cause. The fact that the patient did not have congestive failure removes another possible factor that might have been contributing to the very acute illness.

As far as certainties are concerned, I can say that this patient had rheumatic heart disease with mitral stenosis and auricular fibrillation and emboli to the arterial tree with involvement of both the large arteries of the leg. She had a retroperitoneal hematoma, probably surgical in origin. Coming to less certain things, I think that one must consider myocardial infarction as the most important possibility. I have no proof, especially without an electrocardiogram. Another possibility is that of a final hemorrhage from the site that produced the first hemorrhage.

Dr. Donald S. King: Were any calcium determinations done or calcium therapy given?

Dr. Mallory: I think not. Dr. Linton, you operated on this patient. Do you want to tell us the opinion on the ward?

Dr. Linton: I think first of all that the patient's physician, Dr. Arthur Rappeport, of Quincy, should be complimented on making an early diagnosis and sending the patient to the hospital. As a surgeon, I see a good many cases of peripheral embolism, and unfortunately in the majority of them the patient has been allowed to wait around until gangrene, or practically gangrene, has developed before we have a chance to do an embolectomy. I think every effort ought to be made to teach physicians that there are treatments for peripheral vascular occlusion, such as arterial embolectomy, that are often successful in preventing gangrene; but even if one is not going to do an embolectomy, the medical or conservative methods must be instituted within a few hours. We were very pleased to think that this patient was seen so early and that we had removed so many emboli. I do not recall ever having seen quite so many in any one person. We found them at the bifurcation of the aorta, bifurcation of the common iliac artery and bifurcation of the left femoral artery, all of which were removed. I knew I had left one in the left popliteal artery because I had decided previously that that is not a good site from which to remove emboli. We decided after the operation that the patient had lost about 200 cc. of blood and for that reason only gave her blood replacement of 200 cc.; this was in consultation with Dr. Gordon S. Myers, the cardiologist. When I left the patient about midnight, I thought she was in excellent condition, and I was very much disturbed the following morning to find her blood pressure down to the shock level. I immediately incriminated all of us for using only a 200-cc. blood transfusion, thinking this was the cause of the fall in blood pressure. It became obvious as we watched her that it was not due to blood loss in spite of the fact that we did drain a considerable hematoma from her left flank, because we replaced the blood volume and yet the blood pressure did not return to normal. The urinary output disappeared entirely, and it is of interest that we catheterized her before we did the embolectomy, but unfortunately we did not examine the urine, naturally thinking she would have more urine, but I wish we had now. We believed at first that the oliguria was the result of the low blood pressure, but it became increasingly obvious that some other factor must have been playing a role. It was my opinion that bilateral embolism of the renal artery was unheard of on top of the other emboli that she had had. I was totally at a loss to explain the oliguria. I do not think anybody else had a good explanation for it. I rather tended to think it was the fact that the blood pressure had been low for so long.

Dr. Mallory: Any patient in shock as long as this is a good candidate for lower-nephron nephrosis.

Dr. Linton: That is right. Why we did not get the blood pressure back to normal was never clear to us.

A Physician: Was the adrenal gland implicated in any way?

Dr. Linton: I do not think so. I was a little worried about whether I had damaged the ureters, since I used a retroperitoneal exposure and had really stretched the left ureter. I wondered whether I might have ruptured the left ureter without recognizing it, but this would not explain why the right kidney was not working. I wondered whether she only had one kidney. I did not think we were close enough to the adrenal glands to interfere with them — certainly not the right one.

A Physician: Did heparin account for most of the postoperative hemorrhage?

Dr. Linton: I think it accounted for all of it. The operative site was very dry when we got through. It makes one wonder whether heparin is excreted by the kidneys. It appears from this case as though perhaps some of it is, since she received only the usual dosage of heparin, which ordinarily would keep the clotting time at about twenty minutes, and yet hers in the presence of oliguria rose to over two hours.

Clinical Diagnoses

Rheumatic heart disease, with mitral stenosis.
Arterial embolization to bifurcation of aorta.

Dr. Williams's Diagnoses

Rheumatic heart disease.
Mitral stenosis.
Auricular fibrillation.
Emboli to aorta, and right and left common iliac arteries.
Myocardial infarction.
Anuria due to shock, emboli to kidneys?

Anatomical Diagnoses

Rheumatic heart disease.
Endocarditis, chronic rheumatic, mitral, aortic and tricuspid valves, with mitral stenosis.
Thrombus, left auricular appendage.
Multiple arterial emboli to bifurcation of aorta and common iliac, renal, splenic and left femoral arteries.
Renal infarcts, massive.
Splenic infarcts, recent.
Operations: recent embolectomy, bifurcation of aorta and common iliac and left femoral arteries.

Pathological Discussion

Dr. Mallory: Autopsy showed that this patient had, as was predicted, a severe old mitral endocarditis, with both stenosis and regurgitation. There was relatively minor involvement of the aortic and tricuspid valves as well. The coronary arteries were wide and capacious, and there was no myocardial infarction. One small embolus was found impacted in the foramen ovale, which had a wide mouth on the left side and a very narrow mouth on the right. If this opening had been a little larger, we would have had a paradoxical embolus into the lung, but there were no pulmonary infarcts. Besides the numerous emboli that Dr. Linton found at operation and has already described, there had been numerous others, including several to the spleen, that had caused multiple infarcts and multiple emboli to the renal arteries causing virtually total infarction of both kidneys. There were some old infarcts in the kidney, with fresh massive infarction on each side. There was also evidence of partial infarction of the uterus and rectum and probably a portion of the bladder. The source of all these emboli was thrombosis in the left auricular appendage.

The retroperitoneal tissues of the entire left side of the abdomen, including the left leaf of the diaphragm and all the pelvic tissues, were massively filled with hematoma averaging 5 cm. in thickness and aggregating in all well over 2 liters — nearly 3 liters — of blood. It was not possible to say whether this hemorrhage had all occurred at one time or whether it had occurred in successive episodes. The sutures in the various vessels were all quite tight, and it is unlikely that the bleeding was from them. I believe it occurred diffusely from all the injured tissues in the various operative areas and was consequent to the greatly delayed clotting time.

Dr. Howard B. Sprague: Do you think it is a fair assumption that when, as Dr. Linton says, he took out a great many emboli at one time, in a way a certain log jam had been broken in the auricle and therefore we should suspect that there were a lot more?

Dr. Mallory: I see no way of predicting whether an embolus when it is freed will come away as one solid mass or as fifteen or twenty small ones, which is what I think happened in this case.

Dr. Sprague: I wonder how you can correlate the size of a single solitary embolus with the size of the mitral orifice. How large a one can go through in relation to a particular mitral valve?

Dr. Mallory: This mitral valve was not extremely stenotic. The valve walls were rigid and partially calcified, and the cords very much thickened and shortened. Our impression was that perhaps mitral insufficiency had been a more important factor than stenosis, although some stenosis was present. Certainly, an embolus large enough to occlude either iliac artery could readily have passed through this valve. Whether one large enough to occlude this entire abdominal aorta could have passed, I am not sure.

Dr. Williams: Is it not true that massive infarction of one or both kidneys is often a painless and silent process, or do you believe that such a process is usually accompanied by pain?

Dr. Mallory: I cannot give you figures on that. We have seen them both ways; they may be silent or associated with such marked pain as to lead occasionally to surgical exploration.

CASE 36082

Presentation of Case

A sixty-one-year-old barber was admitted to the hospital because of severe epigastric pain.

Eleven years before the final admission, the patient was first seen in the Out Patient Department complaining of postprandial pain relieved by sodium bicarbonate. Gastritis and an active duodenal ulcer were demonstrated by radiography. Examination of the heart was not remarkable. The blood pressure was 140 systolic, 90 diastolic. One year later he returned with an exacerbation of epigastric distress and was placed on medical therapy. In addition, at that time he complained of pain in the calves on walking. Dorsalis-pedis and posterior-tibial pulses were absent. The feet were cold, and the tips of the great toes were described as dusky. Radiograms of the ankles failed to reveal any arteriosclerosis. Nine years before the last admission the patient entered the hospital for repair of a right indirect inguinal hernia. The

blood pressure was 150 systolic, 80 diastolic. Radiograms revealed cardiac enlargement, chiefly left ventricular, and a tortuous aorta. Three months after herniorrhaphy he returned to the Out Patient Department with substernal pain, which was worse at night and was relieved promptly by nitroglycerin. Some epigastric discomfort was present; radiograms again demonstrated an active duodenal ulcer, as well as a small hiatus hernia. The intermittent claudication was severe enough to prevent walking for any distance (over 200 yards). An electrocardiogram revealed coronary-artery disease and left ventricular strain. He was readjusted on cardiac, dietary and Buerger's-disease regimens. An exacerbation of a gonococcal infection was treated over a period of months in the Out Patient Department. During the past few years there were repeated emotional upsets accompanied by ulcer symptoms, which increased in severity. However, the patient was recalcitrant in following the dietary regimen, continued to smoke 10 to 12 cigarettes per day and indulged in alcohol to excess. Gradually, the anginal pain decreased in severity. Two years before the final entry, note was made that he had not had chest pain for some time; however, he continued to take nitroglycerin. A gastrointestinal series at that time revealed marked deformity of the first portion of the duodenum, with a pseudodiverticulum on the inner-curvature side of the base just distal to which narrowing was evident. Gastritis and duodenitis were present. Radiograms of the lower legs revealed considerable vascular calcification. Sixteen months before the final admission he was treated at another hospital for a posterior myocardial infarction. Recovery from this episode was apparently uneventful. The claudication increased gradually. His final entry into the hospital followed a celebration with much rich food and alcohol two weeks prior to admission; the classic ulcer symptoms had been severe for one week, and for four days he had vomited all food. There had been no hematemesis, melena, weight loss, anorexia or constipation.

Physical examination revealed a well developed, well nourished man with frequent hiccups. The heart was not enlarged; no murmurs were present. The chest was emphysematous. Moderate abdominal distention was present, but there was no epigastric tenderness or palpably enlarged organs. The prostate was moderately enlarged and smooth.

The temperature, pulse and respirations were normal. The blood pressure was 105 systolic, 80 diastolic.

Examination of the blood showed a hemoglobin of 16 gm. and a white-cell count of 9850, with 77 per cent neutrophils. The serum nonprotein nitrogen was 44 mg., and the sugar 114 mg. per 100 cc.; the sodium was 127.7 milliequiv., the chloride 88 milliequiv., the carbon dioxide 32.9 milliequiv., and the potassium 3.6 milliequiv. per liter. The total protein was 7.65 gm. per 100 cc., with an albumin of 4.37 gm. and a globulin of 3.28 gm. (albumin-globulin ratio of 1.3). A twelve-hour gastric specimen revealed 15 units of free hydrochloric acid and a total acid of 26 units. An electrocardiogram revealed a normal rhythm with a rate of 80, left-bundle-branch block, sagging ST segments in Leads AVL, V_5 and V_6, upright T waves in Leads 2 and 3, AVR, F, V_1 and V_2, and inverted T waves in Leads 1, AVL, V_4, V_5 and V_6. Radiograms of the chest disclosed a prominent left ventricle and tortuous aorta. A gastrointestinal series demonstrated prominence of the rugal folds along the greater curvature. After considerable delay barium entered a markedly deformed duodenal bulb, which presented pouches in its proximal portion and only a narrow channel in its distal portion. The mucosa of the duodenum was markedly swollen.

Gastric suction was instituted, and the patient was fed by tube. On the second hospital day some fluid began to pass the pylorus. Sedatives, antispasmodics and intravenous injections of fluids were also given. In spite of diminishing gastric residua, the patient continued to complain of epigastric pain. On the twenty-fourth hospital day, the temperature rose to 102°F., and the white-cell count to 15,000, and he complained of left-lower-quadrant pain, which was a discomfort rather than actual pain. The temperature dropped on the next day, but the white-cell count was 18,800. A barium enema on this day revealed, in spite of inadequate preparation, a 4-cm. area of marked mucosal irregularity at the descending colon-sigmoid junction with what appeared to be a number of nodular masses protruding into the lumen (Fig. 1). A repeat examination two days later showed narrowing in the same area, and considerable difficulty was experienced in guiding the barium stream through this region; however, the area in question could be dilated to almost normal size. Some ir-

regularity of the mucosal pattern was evident in the lower colon and sigmoid. Following the initial portion of this last fluoroscopic examination, the patient was sent from the room to evacuate. He was absent somewhat longer than usual and on his return complained that his legs were numb, weak and partly paralyzed. Superficial examination of the leg reflexes revealed no abnormality. The radial pulse was weak, but apparently regular in

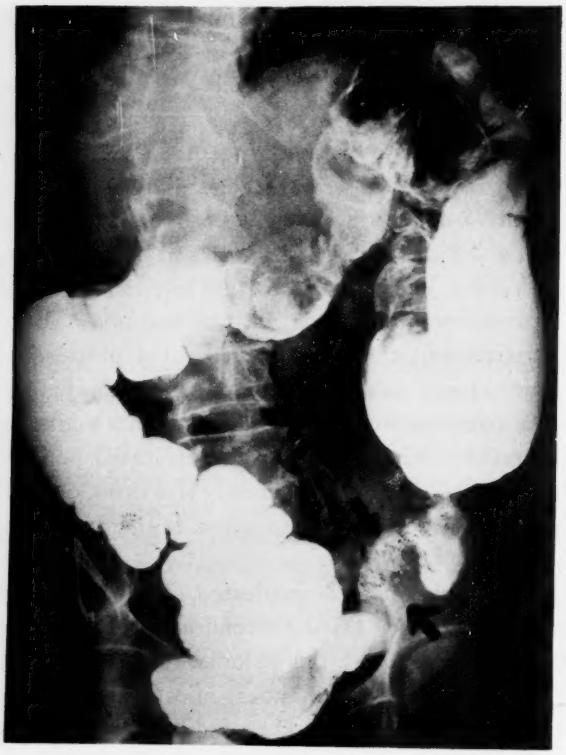

FIGURE 1.

rate and rhythm. No dyspnea or other sign was evident. (Cyanosis, if present, would not have been evident in the fluoroscopy room.) Further fluoroscopy showed that the patient had not evacuated; all the barium remained in the intestines. He was immediately returned to the ward, where, on arrival, he was cyanotic and gasping for breath. Oxygen and tourniquets were applied, but he died within a few minutes on the twenty-sixth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. ROBERT R. LINTON*: This is a long story of a sixty-one-year-old man who obviously had certain diseases that are readily diagnosed and were recognized during his various hospital admissions. We know that he had a duodenal ulcer of eleven years' duration, and I believe it was the reason for admission to the hospital on his last entry. At that time the ulcer had apparently produced sufficient occlusion to the lumen of the duodenum to cause pyloric obstruction. However, I do not think that the duodenal ulcer had anything to do with his death. Anybody who dies as suddenly as he did must almost of necessity have died of some vascular lesion. It was noted ten years previously that he had intermittent claudication as evidenced by the absence of pulses in his feet. Unfortunately, we do not have any other recordings of the pulses in his lower extremities. It is of interest that ten years before admission, which places him at fifty-one years of age, he had no calcification in the blood vessels and therefore somebody might say that he had Buerger's disease. I think this is very unlikely. A man of this age usually has peripheral arteriosclerosis rather than thromboangiitis obliterans as the cause of intermittent claudication.

The patient had had a hernia repaired nine years previously and apparently had no ill effects from this operation although three months later he developed what was thought to be coronary-artery disease requiring nitroglycerin. This apparently was kept under control. Whether his alcoholic intake helped or not I am not competent to say, but I think it probably did not hurt the heart disease. He had a gonococcal infection, which probably had nothing to do with his present illness. It is interesting again that nine years before the final entry his chief complaint was intermittent claudication.

The final admission was chiefly because of an exacerbation of the ulcer symptoms. The abdominal examination was notable because of the lack of any lesion that could be made out. It is interesting that the blood pressure had dropped from 150 systolic (the initial level eleven years previously) to 105 systolic. All the blood chemical findings, I believe, were perfectly consistent with an obstructed pylorus associated with vomiting—including the elevated nonprotein nitrogen and the decrease in the sodium and chloride levels.

DR. TRACY B. MALLORY: I find one note, Dr. Linton, made seven years previously that no pulsation was felt in the leg vessels below the popliteal artery.

*Visiting surgeon, Massachusetts General Hospital.

DR. LINTON: Was there no note during the last episode about the leg vessels?

A PHYSICIAN: The patient did not have any pulses in the legs, and it was questionable whether there was a femoral pulse at the time of the last admission.

DR. LINTON: That observation bears out my opinion that further studies of the arteries of his lower extremities would not help me very much in the diagnosis here since they could not be palpated.

I presume a barium-enema examination was done because he complained of pain in the left lower quadrant, but I am interested in seeing what these x-ray films show.

DR. STANLEY M. WYMAN: The films at hand, Dr. Linton, are those of the last admission and consist of an upper gastrointestinal series and three barium-enema studies. The interesting thing about the gastrointestinal series is the very abnormal pattern in the distal portion of the duodenum and proximal jejunum, with very wide prominent folds. There is a classic deformity of the duodenum consistent with ulcer. There is some prominence of the gastric rugae. The three barium-enema examinations done within a very short time show essentially the same findings — namely, a narrowing in the sigmoid of rather long extent with, on one film, a suggestion of ulceration in the distal sigmoid (Fig 1). The mucosa appears preserved in the proximal sigmoid but cannot be adequately evaluated in the distal sigmoid. The lesion appears much too long to be carcinoma. I think there is little else that can be said about it.

DR. LINTON: Do you have an x-ray film of the chest?

DR. WYMAN: The film is essentially as described, showing a hypertensive, arteriosclerotic type of heart.

DR. LINTON: As I expected these x-ray films are not very helpful to me in making a diagnosis.

It is of some interest that on the twenty-fourth day after admission the patient developed fever for the first time and a leukocytosis and, associated with it, pain in the left lower quadrant. It is my opinion that the gastrointestinal tract does not explain the sudden death of this patient following the barium enema. One always thinks of the worst when dealing with barium enemas. Could the enema have ruptured the colon and suddenly flooded the peritoneal cavity with intestinal contents? Obviously it did not, from an examination of the x-ray films. Furthermore, I am sure that he would not have died quite so suddenly under these conditions.

Could he have died from an air embolus? Having witnessed air embolism in various conditions and seen patients suddenly lose consciousness, I considered that possibility, but I can find no reason why he should have had an air embolus from a barium enema. I do not suppose there could have been anything noxious in the barium enema. I have had 1 patient who died following a magnesium sulfate enema. Apparently the patient (it was a young child) absorbed enough magnesium to poison him so that he died before it was recognized. Since then we have saved a number of patients who have had magnesium poisoning from magnesium sulfate enemas. They are now banned in this hospital and quite rightly so.

To go back to my first premise, it is my opinion that this patient died of a vascular accident. The statement that following the enema his legs felt weak, numb and partly paralyzed certainly shows that something had happened to their main arterial supply. I have no reason to assume that he had an aortic embolism. I do not think aortic embolism is itself a cause for such a sudden death. I would consider the possibility of an acute pancreatitis — left-lower-quadrant pain is sometimes seen associated with pancreatitis, and the widening of the duodenal loop here as shown in the x-ray films suggests some pancreatic lesion, but again I cannot see why he should die so suddenly from any condition involving his pancreas.

I believe therefore that the cause of death was probably the rupture of some part of the cardiovascular system, possibly the heart, although I have no reason to suspect it particularly. He possibly had a dissecting aneurysm that had gone unrecognized. He may have had a rupture of the aorta. Certainly, he had very marked arteriosclerotic disease because that is the one thing that goes through his whole record here. I think I will have to stop there. I cannot go any farther and say at what particular point rupture occurred.

DR. GORDON A. DONALDSON: I should like to ask Dr. Wyman if the picture that one sees in the colon is consistent with thrombosis of the mesenteric vessels.

DR. WYMAN: We have seen very few cases on which to base our opinion, but this picture is not incompatible with it. We did not think of it at the time we saw these films.

DR. JAMES H. CURRENS: Had this patient evacuated most of the barium at the time that he developed his symptoms?

DR. WYMAN: I believe the last film was taken before the patient went to evacuate his enema. He returned having evacuated none, and there is no film to record that. He was rushed upstairs.

CLINICAL DIAGNOSES

Obstructing duodenal ulcer.
Coronary-artery disease.
Dissecting aneurysm? pulmonary embolus? coronary occlusion?

DR. LINTON'S DIAGNOSES

Generalized arteriosclerosis, with obliteration of lower-extremity arteries.
Rupture of aorta.
Duodenal ulcer.

ANATOMICAL DIAGNOSES

Arteriosclerosis, generalized, severe.
Thrombotic occlusion of iliac arteries and abdominal aorta, with occlusion of mouth of inferior mesenteric artery.
Gangrene of sigmoid.
Acute enteritis, focal.
Pulmonary edema, massive.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Autopsy showed that this man did have extensive arteriosclerosis. There was severe coronary sclerosis, with marked narrowing of all the coronary arteries but no complete obstruction of any of the branches. The myocardium showed many patchy fibrous scars but no large area of infarction, and there were no intracardiac thrombi. Both iliac vessels were markedly sclerotic and were filled with old organized thrombus. This thrombus had extended retrogradely up the aorta virtually to, but just below, the level of the renal arteries. It did cover the mouth of the inferior mesenteric artery. There was no rupture of the aorta, but the thrombus found at autopsy was totally occlusive from the level of the internal mesenteric artery downward; part of it was old and well organized, and the central portions were fresh. Our guess was that the final complete obliteration of the abdominal aorta put a sufficient strain upon the heart to result in death from acute left-sided heart failure. The lungs bore that out.

They weighed 1800 gm., which indicates a massive pulmonary edema.

The gastrointestinal tract was interesting. There was an old, scarred ulcer of the duodenum, with a marked pyloric stenosis, and in the large bowel many small areas of acute ulceration were found and one area of complete gangrene just at the junction of the descending colon and sigmoid. Certainly, this area of complete necrosis can be considered infarction based on occlusion of the mesenteric artery. Whether all the small ulcers can be so explained or not I am not sure. There was a good deal of inflammatory infiltration of the mesenteric fat about the bowel, especially in the gangrenous area, which Dr. Reeves, who did the autopsy, believed would account for the narrowing of the bowel that was observed radiologically.

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A NEW SONG

THE American Medical Association, in the report of its National Education Campaign of 1949, refers to the fees paid for membership in various typical organizations of a different nature. These range from $35 collected by the executive leaders of the purchasing agents, through the $45 assessment on architects and the $75 annual levy of the local teamsters unions to the $150 required from the American retail jewelers. Political, educational and other campaigns come extra and come high. It is made apparent that the physicians of the country are being called upon for a small contribution compared with the organizations listed, although no statement appears regarding state and local societies or special colleges, academies and boards for the support of which these laborers in their particular vineyards may be liable.

It is unfortunate that there should seem to be a necessity for any comparison between the American Medical Association and some of the groups mentioned — between a professional association established for service to the people, and the organizations of the motion-picture operators, the purchasing agents and the teamsters, worthy as these fraternities may be. They are organized, it happens, for quite a different purpose.

Educational programs — program is a better word than campaign, which implies a special effort eventually to be concluded — are a necessary part of modern life. They are concerned with that perpetual requirement of gregarious living on which so much emphasis is being placed in these present piping times — better human relations.

Educational programs, however, must be consistent. They must tell, if not the whole truth, at least a large part of it, and no untruths. They must reveal the faults in human conduct as well as its virtues; its failures as well as its successes.

Satisfactory human relations have always been difficult but rarely impossible to attain. They have been the reward of a superior degree of candor, an ability to see all sides of a controversial matter and the capacity for free and friendly discussion regarding it.

There is much ground to cover if people are to become even a little better adjusted to each other than at present, for they are living in a period of more than usual strain and stress. They are living in the midst of increased racial and religious and other social prejudices, and of economic and political uncertainties.

The profession of medicine, with so much in its record to recommend it, has nevertheless much that can be improved in both the quality and the distribution of its service. The more that is told of its story and the greater readiness there is to establish mutual understandings, the better will relations become. The second half of the century must be more productive in this respect than was the first.

A recurrent theme of the Psalms of David — "I will sing a new song unto thee" — might also represent a turning point in the public relations of a profession.

DECORUM DECANORUM

At the recent American Conference of Academic Deans, serious charges were made against those responsible for medical education. Some of the remarks about their fellow educators were couched in language that must have shocked that presumably dignified and decorous gathering. Dean Simeon E. Leland, of Northwestern University, charged that the medical schools were using "Petrillo and Fishbein Economics" in admitting students, implying that the profession was deliberately holding down the number of new admissions to its membership in its own selfish interest. "We need both more and better doctors" declared Dean Leland, as quoted in the *New York Times*. "If we had more, we would have better doctors. There would be more opportunity for medical research, and competition would weed out the weaker ones as it does in the other professions."

Dean William S. Guthrie, of Ohio State University, pointed out that in 1905 there were 160 medical schools in this country, graduating a total of 5606 students, whereas in 1949, 79 medical schools graduated only 5094. He deplored the fact that "No matter how eligible, three-fourths of the applicants just can't get in" and urged a return to the accelerated program of the war years.

Dr. Joseph C. Hinsey, president of the Association of American Medical Colleges and dean of Cornell University Medical College, was quick to come to the defense of the medical schools in letters to the *Times* and the *Herald-Tribune*. He referred to Dr. Victor Johnson's figures, which indicate that there is at present an annual excess of medical graduates over deaths of physicians of about 2000, and pointed out that since the beginning of World War II, seven new four-year medical schools have come into being in the United States and five others are in prospect or contemplated. Expansion of existing schools will further increase the annual increment of physicians.

The point of view of the deans appears to be that the medical schools should expand their facilities to take care of all qualified applicants, rather than in accordance with the need of the populace for medical care. Fortunately, there are countries in Europe that furnish a demonstration of the effects of just such a policy. In Germany, in Austria and in Italy there are medical schools with a thousand or more students in a class. All qualified observers, and indeed the faculties of these very schools, are agreed that the quality of medical teaching has suffered greatly as a result of such overcrowding. More serious still has been the overproduction of medical graduates in these countries since the war, for, contrary to Dean Leland's naïve notion, cutthroat competition in an overmanned medical profession will not necessarily cause the best physicians to be the most successful. Such conditions generate impelling pressures to resort to unscrupulous practices for survival and lead inevitably to deterioration of ethical standards.

What is to be done about the 15,000 applicants who are denied admission to medical schools every year? Why does this represent such an appalling aggregate of frustration and broken hopes? It is largely due to the fact that in most colleges the student who is preparing for medicine is set apart and classified as such. Certain "premedical" courses are prescribed. He joins a "premedical" club. He identifies himself in his own mind and in the minds of his family and friends with a medical career long before he has demonstrated an aptitude for such a calling. When the door to medical school is not opened to him, after he has publicly committed himself to it, it is as if it had been slammed in his face. He is humiliated. He finds himself unprepared both psychologically and by virtue of his specialized preparation to accept any alternative.

College administrators could alleviate much of this distress if they abandoned the term "premedical student" and took pains to prepare every prospective medical student for an alternative career if medicine were not open to him.

They should realize that the larger interest of society would be best served by the gearing of the production of doctors to the demands for medical service rather than to the desires of candidates for medical schools.

If anything has been gained from this name calling, it has been to bring into focus more clearly than ever the pressing need for an impartial inquiry into the medical manpower requirements of the United States and how they are being met.

POSTGRADUATE LECTURE COURSE

THE program of the fifth postgraduate lecture course to be arranged by the Committee on Postgraduate Medical Education of the Massachusetts Medical Society appears elsewhere in this issue of the *Journal*. It is to be noted that the course will be given this year in Ware Hall at the Boston Medical Library instead of in Sanders Theater in Cambridge.

Originated as the Society's contribution to the further education of returning veterans, the course achieved an immediate popularity that made its perennial repetition almost an obligation. It is a source of satisfaction that this should be so, for it gives further strength to the conviction that postgraduate education is one of the permanent functions of a medical society. Such education, moreover, should be extended beyond the membership of the Society itself, as is the case with this postgraduate lecture course, which is open to all physicians and medical students who wish to register.

Fourteen sessions will be held, beginning on March 6 and ending on May 1, the Monday sessions starting at 7 and ending at 9 p.m., the Wednesday sessions taking place in the afternoon between 4 and 6 p.m. The instructors are all experts in their fields, and the subjects that have been chosen are timely, interesting and instructive.

WHITE HOUSE CONFERENCE

PLANS for the Midcentury White House Conference on Children and Youth, to be held in Washington during the week of December 3, 1950, are off to a flying start, according to the *Progress Bulletin* of the Conference. Members of the National Committee of the Conference agree with President Truman that "the greatest challenge facing the world today is how it can help its children to be secure in themselves, in their families and in their communities." To meet this challenge and its nationwide response, it has been necessary to extend the plans and activities for the Conference on all fronts.

This year, Katharine F. Lenroot, chief of the Children's Bureau and secretary of the National Committee, has announced that "the accent on mental, emotional and spiritual development as the focus of the Conference's concern for children and youth has struck a responsive chord in layman and professional worker alike."

Already, according to plans previously made public by Mr. Oscar R. Ewing, White House Conference chairman, representatives of approximately 600 national organizations with interests in children and youth have met in Washington to make specific arrangements for participation. Youth itself, furthermore, will take part in this particular conference on children and youth although "surprisingly this will be the first of the decennial White House Conferences on Children and Youth in which young people will take active part."

As organization of the Conference progresses and committees are designated in state after state, its leaders may indeed be grateful for the painstaking and thorough study of the nation's child health services that has so recently been completed by the American Academy of Pediatrics.

Dr. T. S. Lambert . . . recently petitioned the legislature for leave to lecture before that body. Firstly, on the evils of quackery and quack medicines, and the importance that something be done to check the impositions from which the public suffers so much at present. Secondly, to show that life commences at the time of conception, and that abortion cannot be procured at any time without high crime, as it respects the child, and great injury to the mother.

Boston M. & S. J., *February 20, 1850*

Fourth-year students in approved medical schools are reminded that the *Journal's* prize-essay contest on "Recent Advances in the Recognition, Treatment and Control of Poliomyelitis" closes on March 15. Manuscripts must be in the hands of the editor on that date.

For further information see *The New England Journal of Medicine*, September 29, 1949, p. 506, or write to the editor.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

HARLOW — John S. Harlow, M.D., of Boston, died on February 6. He was in his fifty-fourth year.

Dr. Harlow received his degree from Harvard Medical School in 1923.

Two sisters survive.

POSTGRADUATE LECTURE COURSE

The fifth Postgraduate Lecture Course, which has been arranged by the Committee on Postgraduate Medical Education of the Massachusetts Medical Society, in co-operation with the Massachusetts Department of Public Health, will begin on March 6. The meetings will be held in John Ware Hall, Boston Medical Library, 8 Fenway, Boston. These lectures are designed for all physicians of Massachusetts, and surrounding states, medical officers, hospital residents, interns, medical students and postgraduate students. Registration is free, and membership in the Massachusetts Medical Society is not required for admission.

All those who plan to attend should enroll immediately by addressing a post card or letter directly to the Postgraduate Lecture Course Committee, Massachusetts Medical Society, 8 Fenway, Boston 15 (telephone, COmmonwealth 6-2800.

The detailed program of the course, which will be given in the evenings on Mondays and the afternoons on Wednesdays, is as follows:

Monday, March 6. RHEUMATISM AND ALLIED DISEASES. Chairmen: Theodore B. Bayles and J. Sydney Stillman.

7:00–7:30. **The Physiology of the Adrenal-Cortical Steroids.** Peter H. Forsham, instructor in medicine, Harvard Medical School, and junior associate in medicine, Peter Bent Brigham Hospital.

7:30–8:00. **Observations on the Experimental Treatment of Rheumatoid Arthritis with ACTH and Cortisone.** Theodore B. Bayles, clinical associate in medicine, Harvard Medical School, visiting physician and director of research, Robert Breck Brigham Hospital, and associate in medicine, Peter Bent Brigham Hospital.

8:00–8:20. **ACTH in Rheumatic Fever and Rheumatic Carditis.** Benedict F. Massell, clinical associate in pediatrics, Harvard Medical School, chief, Rheumatic Fever Division, Children's Hospital, and associate research director, House of the Good Samaritan.

8:20–8:40. **Observations on ACTH and Cortisone in Juvenile Rheumatoid Arthritis** (with motion pictures). J. Sydney Stillman, assistant in medicine, Harvard Medical School, and chief, Medical Service, Robert Breck Brigham Hospital.

8:40–9:00. **Panel Question Period.**

Wednesday, March 8. CARDIOVASCULAR DISEASE. Chairmen: C. Sidney Burwell and David I. Rutledge.

4:00–4:20. **Prevention of Recurrences of Acute Rheumatic Fever.** George P. Sturgis, visiting physician, Massachusetts General Hospital, and visiting physician and research associate, House of the Good Samaritan.

4:20–4:30. **Discussion.**

4:30–4:50. **Iatrogenic Cardiac Disease.** Hugh P. Greeley, physician, Department of Internal Medicine, Lahey Clinic.

4:50–5:00. **Discussion.**

5:00–5:20. **Water and Salt in Heart Disease.** Samuel H. Proger, physician-in-chief, New England Center Hospital, chief of staff, Joseph H. Pratt Diagnostic Hospital, and professor of medicine, Tufts College Medical School.

5:20–5:30. **Discussion.**

5:30–5:50. **Pitfalls in Electrocardiography.** Conger Williams, associate physician, Massachusetts General Hospital.

5:30–6:00. **Discussion.**

Monday, March 13. CARDIOVASCULAR DISEASE. Chairmen: C. Sidney Burwell and David I. Rutledge.

7:00–7:20. **The Differential Diagnosis of Cardiac Pain.** Herrman L. Blumgart, professor of medicine, Harvard Medical School, and physician-in-chief, Beth Israel Hospital.

7:20–7:30. **Discussion.**

7:30–7:50. **Physiological Considerations of Practical Value in the Treatment of Peripheral Vascular Disease.** Robert W. Wilkins, associate professor of medicine, Boston University School of Medicine, and assistant director, Evans Memorial, Massachusetts Memorial Hospitals.

7:50–8:00. **Discussion.**

8:00–8:20. **The Management of Heart Disease during Pregnancy.** Burton E. Hamilton, consultant in cardiology, Boston Lying-in Hospital.

8:20–8:30. **Discussion.**

8:30–8:50. **The Cardiac Patient As a Surgical Risk.** David I. Rutledge, physician, Department of Internal Medicine, Lahey Clinic.

8:50–9:00. **Discussion.**

Wednesday, March 15. GYNECOLOGY. Chairmen: Paul A. Younge and Christopher J. Duncan.

4:00–4:20. **Trends in Gynecological Surgery.** Christopher J. Duncan, instructor in gynecology, Harvard Medical School, visiting surgeon, Free Hospital for Women, and assistant in obstetrics, Tufts College Medical School.

4:20–4:30. **Discussion.**

4:30–4:50. **The Treatment of Dysmenorrhea.** Francis M. Ingersoll, instructor in gynecology, Harvard Medical School, and assistant in surgery, Massachusetts General Hospital.

4:50–5:00. **Discussion.**

5:00–5:20. **Clinical Use of Hormones in Gynecology.** William J. Mulligan, assistant in gynecology and obstetrics, Harvard Medical School, assistant obstetrician to outpatients, Boston Lying-in Hospital, and surgeon to outpatients, Endocrine Clinic, Free Hospital for Women.

5:20–5:30. **Discussion.**

5:30–5:50. **Modern Concepts in the Early Diagnosis and Treatment of Carcinoma in Situ of the Cervix.** Paul A. Younge, instructor in gynecology, Harvard Medical School, visiting surgeon, Free Hospital for Women, and surgeon, New England Deaconess Hospital.

5:50–6:00. **Discussion.**

Monday, March 20. NO LECTURE.

Wednesday, March 22. INFECTIOUS DISEASES AND ANTIBIOTIC THERAPY. Chairmen: Maxwell Finland and Louis Weinstein.

4:00–4:20. **Use of New Antibiotics in Infants and Children.** Harry Shwachman, associate in pediatrics, Harvard Medical School, and chief, Division of Clinical Laboratories, Children's Hospital.

4:20–4:35. **Antibiotics in Pneumonia.** Maxwell Finland, physician-in-chief, Fourth Medical Service, Boston City Hospital, and assistant professor of medicine, Harvard Medical School.

4:35–4:50. **Allergic Manifestations from Antibiotics.** Chester S. Keefer, Wade Professor of Medicine, Boston University School of Medicine, physician-in-chief, Massachusetts Memorial Hospitals, and director, Evans Memorial.

4:50–5:05. **Discussion on Antibiotics.**

5:05–5:20. **Diphtheria — Present Status.** Edwin H. Place, physician-in-chief, South Department for Contagious Diseases, Boston City Hospital, and professor of pediatrics, Tufts College Medical School.

5:20–5:35. **Pertussis — Prevention and Treatment.** R. Cannon Eley, associate in pediatrics and communicable diseases, Harvard Medical School, and chief of Isolation Service, Infants and Children, Children's Hospital.

5:35–5:50. **Streptococcal Infections.** Louis Weinstein, chief of service, Department of Infectious Diseases, Haynes Memorial Hospital, associate professor of medicine, Boston University School of Medicine, and lecturer in infectious diseases, Harvard Medical School.

5:50–6:00. **Discussion.**

Monday, March 27. Diabetes. Chairmen: Alexander Marble and David Hurwitz.

7:00–7:20. **Nonpancreatic Factors in Diabetes.** Peter H. Forsham, instructor in medicine, Harvard Medical School, and junior associate physician, Peter Bent Brigham Hospital.

7:20–7:40. **Use of Fibril Elongation as an in Vitro Assay for Insulin.** David F. Waugh, assistant professor of physical biology, Massachusetts Institute of Technology.

7:40–8:05. **Treatment of the Ambulatory Diabetic.** David Hurwitz, clinical associate in medicine, Harvard Medical School, junior visiting physician, Boston City Hospital, visiting physician, Mt. Auburn Hospital, and consultant in diabetes, Boston Lying-in Hospital.

8:05–8:30. **Diabetic Coma.** Alexander Marble, clinical associate in medicine, Harvard Medical School, and physician, New England Deaconess Hospital.

8:30–8:55. **Treatment of Foot Lesions in Diabetes.** Clifford C. Franseen, surgeon, New England Deaconess Hospital.

8:55–9:00. **Question Period.**

Wednesday, March 29. Psychiatry. Chairmen: Robert Fleming and Augustus S. Rose.

4:00–5:00. **Indications for Lobotomy and Electric Shock Treatment.** Augustus S. Rose, instructor in neurology and psychiatry, Harvard Medical School, and associate neurologist, Peter Bent Brigham and Massachusetts General hospitals.

5:00–6:00. **Recent Advances in Therapy of Chronic Alcoholism, including Experience with Antabuse.** Robert Fleming, instructor in psychiatry, Harvard Medical School, and associate in psychiatry, Peter Bent Brigham Hospital.

Monday, April 3. Pediatrics. Chairmen: R. Cannon Eley and Warren R. Sisson.

7:00–7:30. **Treatment with Antibiotics of Pulmonary Tuberculosis in Children.** William Berenberg, associate physician, Children's Hospital, and instructor in pediatrics, Harvard Medical School.

7:30–8:00. **The Use of Antibiotics in Chronic Pulmonary Disease.** Robert M. Smith, anesthesiologist, Children's Hospital.

8:00–8:30. **Relation of Chronic Nasopharyngeal Infection and Pulmonary Infection in Children.** Carlyle G. Flake, chief, Division of Otolaryngology, Children's Hospital, and instructor in laryngology, Harvard Medical School.

8:30–9:00. **Diagnosis and Management of Diphtheria.** Louis Weinstein, chief of service, Department of Infectious Diseases, Haynes Memorial Hospital, associate professor of medicine, Boston University School of Medicine, and lecturer in infectious diseases, Harvard Medical School.

Wednesday, April 5. Anticoagulants and Surgical Therapy in Thromboembolic and Heart Disease. Chairmen: James A. Evans and Robert R. Linton.

4:00–4:20. **The Prophylactic and Surgical Treatment of Thromboembolic Disease.** Gordon A. Donaldson, assistant surgeon, Massachusetts General Hospital, and assistant in surgery, Harvard Medical School.

4:20–4:25. **Question Period.**

4:25–4:30. **Summary of Results in Anticoagulant Treatment of Postoperative Thromboembolic Disease.** James A. Evans, physician, Department of Internal Medicine, Lahey Clinic and New England Baptist and New England Deaconess hospitals.

4:30–4:50. **Basic Physiologic Considerations in the Control of Thromboembolic Disease by Anticoagulants.** Benjamin Alexander, assistant professor of medicine, Harvard Medical School, associate director, Beth Israel Hospital, and staff member, Medical Service, Beth Israel Hospital.

4:50–4:55. **Question Period.**

4:55–5:10. **Laboratory Methods in Control of Dicumarol Treatment.** H. J. Perkins, director of clinical laboratory, Lahey Clinic.

5:10–5:15. **Question Period.**

5:15–5:30. **The Treatment of Peripheral Arterial Emboli.** Richard Warren, assistant in surgery, Massachusetts General Hospital, clinical associate in surgery, Harvard Medical School, consultant in vascular surgery, Veterans Administration Hospital, West Roxbury.

5:30–5:35. **Question Period.**

5:35–5:55. **Cardiac Aspects of Thromboembolic Disease Including Coronary Disease.** Paul D. White, consultant, Massachusetts General Hospital, clinical professor of medicine, Harvard Medical School, and executive director, National Advisory Heart Council.

5:55–6:00. **Question Period.**

Monday, April 10. Bone and Joint Diseases. Chairmen: Joseph S. Barr and William T. Green.

The Hip and the General Practitioner

7:00–7:30. **The Problems of Infancy and Childhood.** Paul W. Hugenberger, instructor in orthopedic surgery, Harvard Medical School, and associate orthopedic surgeon, Children's Hospital.

7:30–7:45. **The Problems of Adolescence.** Armin Klein, instructor in orthopedic surgery, Harvard Medical School, visiting orthopedic surgeon, Massachusetts General Hospital, and head, Department of Orthopedics, Beth Israel Hospital.

7:45–8:10. **The Problems of the Aging Hip.** Otto E. Aufranc, instructor in orthopedic surgery, Harvard Medical School, and assistant orthopedic surgeon, Massachusetts General Hospital.

8:10–8:30. **Fractures of the Hip.** Thomas B. Quigley, clinical associate in surgery, Harvard Medical School and associate in surgery, Peter Bent Brigham Hospital.

8:30–9:00. **Round Table Discussion.**

Wednesday, April 12. Poliomyelitis and Other Acute Central Nervous System Infections. Chairmen: David D. Rutstein and Conrad Wesselhoeft.

4:00–4:20. **General Aspects of Poliomyelitis.** Conrad Wesselhoeft, clinical professor of infectious diseases, Harvard School of Public Health.

4:20–4:40. **Epidemiology of Poliomyelitis and Other Central-Nervous-System Infections.** David D. Rutstein, professor of preventive medicine, Harvard Medical School.

4:40–5:10. **Differential Diagnosis and Treatment of Poliomyelitis.** Louis Weinstein, chief of service, Department of Infectious Diseases, Haynes Memorial Hospital, associate professor of medicine, Boston University School of Medicine, and lecturer in infectious diseases, Harvard Medical School.

5:15–5:30. **Treatment of Poliomyelitis.** R. Cannon Eley, associate in pediatrics and communicable diseases, Harvard Medical School, and chief, Isolation Service, Infants and Children, Children's Hospital.

5:30–5:50. **Phrenic Stimulation in Certain Types of Bulbar Poliomyelitis** (with motion picture). Stanley J. Sarnoff, assistant professor of physiology, Harvard School of Public Health and assistant in medicine, Peter Bent Brigham Hospital.

5:50–6:00. **Question Period.**

Monday, April 17. No Lecture.

Wednesday, April 19. No Lecture.

Monday, April 24. HEMATOLOGY. Chairmen: Frank H. Gardner and William C. Moloney.

7:30–7:35. **Macrocytic Anemias.** Maurice Strauss, clinical associate in medicine, Harvard Medical School, and chief, Medical Service, Cushing Veterans Administration Hospital.

7:35–8:15. **Practical Problems Related to the Rh System.** Fred H. Allen, Jr., instructor in pediatrics, Harvard Medical School, and associate director, Blood Grouping Laboratory, Children's Hospital.

8:15–8:50. **Diagnosis of Hemolytic Anemias.** Frank H. Gardner, instructor in medicine, Harvard Medical School, and associate in medicine, Peter Bent Brigham Hospital.

8:50–9:00. **Question Period.**

Wednesday, April 26. CANCER. Chairmen: Joseph C. Aub and Ira T. Nathanson.

4:00–4:25. **Progress and Pathological Diagnosis of Cancer.** Shields Warren, professor of pathology, Harvard Medical School, and pathologist, New England Deaconess Hospital.

4:25–4:30. **Question Period.**

4:30–4:55. **Progress in the Radiological Diagnosis of Cancer.** Merrill C. Sosman, professor of radiology, Harvard Medical School, and radiologist, Peter Bent Brigham Hospital.

4:55–5:00. **Question Period.**

5:00–5:25. **Principles in the Management of Lymph-Node Metastases.** Grantley W. Taylor, assistant professor of clinical surgery, Harvard Medical School, and visiting surgeon, Massachusetts General Hospital.

5:25–5:30. **Question Period.**

5:30–5:55. **Chemotherapy in Leukemia and Leukosarcoma.** William Dameshek, professor of clinical medicine, Tufts College Medical School, and hematologist, New England Center Hospital.

5:55–6:00. **Question Period.**

Monday, May 1. OBSTETRICS. Chairmen: John L. Newell and Judson A. Smith.

I. BLEEDING IN THE FIRST TRIMESTER OF PREGNANCY.

7:00–7:20. **The Clinical Aspects.** H. Bristol Nelson, senior obstetrician, Boston Lying-in Hospital, and instructor in obstetrics, Harvard Medical School.

7:20–7:40. **The Pathological Aspects.** Joseph Anderson, assistant resident in pathology, Boston Lying-in Hospital.

7:40–8:00. **Endocrinologic Aspects and the Rationale of Hormone Therapy.** William J. Mulligan, assistant in gynecology and obstetrics, Harvard Medical School, assistant obstetrician to outpatients, Boston Lying-in Hospital, and surgeon to outpatients, Endocrine Clinic, Free Hospital for Women.

II. BLEEDING IN THE THIRD TRIMESTER OF PREGNANCY.

8:00–8:20. **The Clinical Aspects of Separation of the Placenta.** Benjamin Tenney, Jr., professor of obstetrics and gynecology, Boston University School of Medicine.

8:20–8:40. **Clinical Aspects of Placenta Previa.** Judson A. Smith, senior obstetrician, Boston Lying-in Hospital, and instructor in obstetrics, Harvard Medical School.

8:40–9:00. **Pathological Aspects and Blood Changes in Premature Separation of the Placenta.** Albert E. Weiner, assistant resident, Boston Lying-in Hospital, and teaching fellow in obstetrics, Harvard Medical School.

NEW HAMPSHIRE MEDICAL SOCIETY

MEDICAL TECHNOLOGY CURRICULUM

A medical technology curriculum, designed to meet a need for professionally trained young women in New Hampshire hospitals, will be offered by the University of New Hampshire next fall. Dr. Lawrence W. Slanetz, professor of bacteriology, will supervise the new curriculum.

The Board of Trustees has approved a program in the College of Liberal Arts that will give more systematic preparation for students now enrolled as majors in biology and bacteriology.

The College has prepared the new curriculum in co-operation with clinical pathologists in hospitals at Manchester, Hanover and Concord who desire to appoint New Hampshire graduates to their staffs. The course leads to a degree of Bachelor of Science.

Included in the curriculum is a twelve-month training period at a hospital approved by the Council on Medical Education and Hospitals of the American Medical Association covering lectures and laboratory work in bacteriology, biochemistry, hematology, histology and serology.

DEATH

HEBERT — Simeon L. Hebert, M.D., of Manchester, died on January 17. He was in his fiftieth year.

Dr. Hebert received his degree from University of Vermont College of Medicine in 1926. He was a member of the staff of Hillsborough County General Hospital and former president of the staff of Notre Dame de Lourdes Hospital.

His widow, his mother, a son, a daughter and a brother survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

The March schedule for Consultation Clinics for Crippled Children in Massachusetts under the provisions of the Social Security Act follows:

ORTHOPEDIC CLINICS	DATE	CLINIC CONSULTANT
Haverhill	March 1	William T. Green
Greenfield	March 2	Charles L. Sturdevant
Lowell	March 3	Albert H. Brewster
Salem	March 6	Paul W. Hugenberger
Brockton	March 9	George W. Van Gorder
Gardner	March 14	Carter R. Rowe
Pittsfield	March 15	Frank A. Slowick
Worcester	March 17	John W. O'Meara
Springfield	March 21	Garry deN. Hough, Jr.
Hyannis	March 23	Paul L. Norton
Fall River	March 27	David S. Grice
RHEUMATIC FEVER CLINICS		DATES
Fitchburg		March 1, 8, 15, 22, 29
North Reading		March 7, 14, 21, 28
PLASTIC CLINICS		CONSULTANT
Eastern Massachusetts Clinics bi-monthly		Bradford Cannon
Western Massachusetts Clinics monthly		Joseph M. Baker

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

BLUE CROSS-BLUE SHIELD

INCREASED BLUE CROSS BENEFITS

Massachusetts Hospital Service, Incorporated, has announced increased benefits for group plan and semi-private members to become effective

March 1, 1950, at no increase in cost. These benefits are as follows:

1. For one year beginning March 1 additional room and board credit of $1 from 7th through 14th, $2 from 15th through 22nd, and $3 from 23rd through 30th day of hospitalization.
2. Full credit for "Included Services" for the 61st through the 120th day.
3. Elimination of maternity waiting periods for newlyweds, *if member in GROUP six months on an individual contract and change to family contract is made within 60 days of date of marriage* — Blue Cross must be notified of date of marriage.
4. Admissions caused by pregnancy but prior to the seventh month will be treated as regular medical-surgical admissions with no waiting period.
5. Waiting period for admission for tonsil and adenoidectomies for adults is eliminated as well as the limitation of the two-day restriction.
6. Out-patient surgery is an added benefit for one year beginning March 1.
7. Admission for congenital anomalies will be honored after a one-year waiting period.
8. Payments for charges for other than room and board accommodations in nonparticipating hospitals will be up to $150, or $5 per day, whichever is greater, for like admissions in participating hospitals.

MISCELLANY

ARMY CIVILIAN INTERN PROGRAM

Selection of senior medical students for the Army's civilian intern program has proceeded at an accelerated rate since the first of this year. To date, 52 applicants have been selected. They represent 34 of the country's approved medical schools and will intern in 48 different civilian hospitals.

The first group of applications was processed on December 13. Since that date the Surgeon General's Selection Board has convened five more times in an effort to keep up with the increasing number of applications being received. It will continue to meet at frequent intervals until it has selected the 300 candidates for whom the program provides spaces.

To be eligible, a candidate must be a potential graduate who will begin an internship between January 1 and December 31, 1950, and must have been accepted for internship training in a civilian hospital acceptable to the Surgeon General.

Successful candidates will be commissioned in the Army Medical Corps Reserve and permitted to complete their internships as officers on active duty with pay and certain allowances of the grade of first lieutenant.

Physicians selected for this training program will serve two years on a duty status for the year or major portion of a year of formal training received.

BOOK REVIEW

Epidemiology in Country Practice. By William Norman Pickles, M.D. (Lond.), medical officer of health, Aysgarth Rural District, with a preface by Major Greenwood, F.R.S., D.Sc., F.R.C.P., professor of epidemiology and vital statistics in the University of London. Re-issued 1949. Cloth, 112 pp., with 17 illustrations including 11 charts. Bristol: John Wright and Sons Ltd. London: Simpkin Marshall Ltd. 1939. 10/6.

As the publishers note, when this charming little book was first published in May, 1939, "it was quickly recognized as being one of those rare books which are destined to become 'classics.'" Unfortunately in April, 1941, its steady and continuous sale was interrupted by the destruction, through enemy action, of the entire stock and type.

In response to numerous appeals the work has finally been reissued in practically its original form. This new issue thus amounts to a republication without revision, which in the case of such a complete essay would have been superfluous.

Those who heard Dr. Pickles's Cutter Lecture on Preventive Medicine delivered at the Harvard School of Public Health in April, 1948, or read it in the *Journal* of September 16 of the same year are already familiar with what amounted to a condensation of this delightful book.

NOTICES

ANNOUNCEMENT

Drs. Richard Schatzki and John E. Gary announce the opening of an office at 1180 Beacon Street, Brookline, for the practice of radiology.

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

A meeting of the Boston City Hospital House Officers' Association will be held in the Dowling Amphitheater of the Hospital on Tuesday, March 7, at 7 p.m. Dr. Chester M. Jones will speak on the subject "Nutritional Problems Arising from Anastomotic Surgery."

NEW ENGLAND CARDIOVASCULAR SOCIETY

A meeting of the New England Cardiovascular Society (formerly, the New England Heart Association) will be held at the George Sherman Auditorium, Beth Israel Hospital, Boston, on March 6, at 8 p.m., Dr. Herrman L. Blumgart presiding.

PROGRAM

The Width of the Azygos Vein — A Roentgenographic Sign of Venous Engorgement. Drs. Felix G. Fleischner and Sanford W. Udis.

Experiences with Protoveratrine, A Pure Veratrum Alkaloid, with Some Clinical Implications. Dr. Edward Meilman.

Hemorrhagic Pericarditis in Dicumarol-Treated Patients with Acute Myocardial Infarction. Drs. Louis Wolff and Robert Goldstein.

The Pathogenesis of Angina Pectoris. Drs. Paul M. Zoll and Stanford Wessler.

Further Experience with Radioactive Iodine in the Treatment of Euthyroid Cardiac Patients with Angina Pectoris and Congestive Heart Failure. Drs. Herrman L. Blumgart, A. Stone Freedberg and George S. Kurland.

Interested physicians and medical students are cordially invited to attend.

NEW ENGLAND HEALTH EDUCATION ASSOCIATION

The spring meeting of the New England Health Education Association will be held in Springfield, Massachusetts, on March 10 and 11. The subject of the meeting will be "Mental Health Education — Its Practical Application in the School and in the Community."

The conference opens at 2 p.m. on Friday and will close early Saturday afternoon. The place and further details will be announced later.

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

The thirty-fifth annual meeting of the American Association of Industrial Physicians and Surgeons and four other industrial associations — the American Industrial Hygiene Association, American Conference of Governmental Industrial Hygienists, American Association of Industrial Nurses and American Association of Industrial Dentists —will be held in the Sherman Hotel, Chicago, April 22–29. The registration during the meeting is expected to exceed 2000.

The various section meetings will deal with general manufacturing, steel and heavy industry, petroleum, rubber and chemicals, hand and restorative surgery, workmen's compensation and insurance, and the mining industry. On Monday afternoon and Tuesday morning, April 24 and 25, there will be clinics at St. Luke's and Cook County hospitals. These clinics will present the newer types of diagnosis and treatment of industrial injuries and the handling of medical cases. The session on Tuesday evening will be devoted to a symposium on alcoholism in industry. All five sponsoring groups will gather at a joint meeting Thursday afternoon. The program will be built around the convention theme "Teamwork in Industrial Health," which is aimed at rendering a comprehensive, complete medical service to all industrial workers. During the conference, more than 20 large industrial plants in the Chicago area will have open house in their medical departments for industrial physicians and surgeons and industrial nurses.

WOMAN'S AUXILIARY TO SUFFOLK DISTRICT MEDICAL SOCIETY

The March meeting of the Woman's Auxiliary to Suffolk District Medical Society will be held in Sprague Hall, Boston Medical Library, on Thursday afternoon, March 2. Dr. William E. Browne, chairman of the Legislative Committee for Suffolk District Medical Society, will speak on the subject "Why Medical Legislation Is Important."

There will be a coffee hour at 1:30, and the business meeting will be held at 2:15.

EASTERN SECTION OF AMERICAN CONGRESS OF PHYSICAL MEDICINE

A meeting of the Eastern Section of the American Congress of Physical Medicine will be held in Washington, D. C., on Saturday, April 29. The afternoon session, starting at 2 p.m., will be conducted at the George Washington University Hospital, 901 23rd Street, North West, and after dinner at the Willard Hotel, the evening session will consist of a panel discussion. Full program will be published when final plans have been completed. Chairman of the Section is Colonel Emmett M. Smith, M.C. Inquiries should be directed to Herman L. Rudolph, M.D., Section Secretary, 400 North Fifth Street, Reading, Pennsylvania.

WORLD FEDERATION FOR MENTAL HEALTH

The third annual meeting of World Federation for Mental Health will be held in Paris, by kind invitation of the Ligue d'Hygiène Mentale, from Thursday, August 31, to Thursday, September 7, 1950, inclusive. (The dates have been changed since publication of the December Bulletin of World Federation for Mental Health.) Meetings will be held in the Cité Universitaire, which is situated south of the Seine, overlooking the Parc de Montsouris, about 3 miles from the Place de la Concorde. The Cité Universitaire is served by the Metro (Underground), and by busses. Membership will comprise the delegation appointed in each country by the convening organization, in conjunction with other member associations where these exist, and, in addition, any other members of member associations who are able to attend the meeting.

The first six days of the meeting will be devoted to the discussion of the following scientific topics primarily in small working groups: mental health in education; occupational and industrial mental health; mental-health problems of transplanted and homeless persons; and problems of leadership and authority in local communities. The administrative sessions will be held on Wednesday and Thursday, September 6 and 7. The sixth meeting of the Executive Board of the Federation will be held from Sunday, August 27 to Wednesday, August 30, and the seventh meeting on Friday and Saturday, September 8 and 9.

A certain number of students' rooms in the Cité Universitaire will be available for those attending the meeting, and will be allocated by the Secretariat of the Federation in rotation, as requests for them are received. (Details of cost and so forth can be supplied by the Division of World Affairs, The National Committee for Mental Hygiene.) Early application should be made to the Secretary of the Federation, 19 Manchester Street, London, W 1, since the number of rooms available will be limited.

Hotel reservations should be made direct or through the usual travel agencies.

TOURS OF DUTY FOR AIR FORCE MEDICAL AND DENTAL RESERVE OFFICERS

A regulation has been published by the United States Air Force permitting medical and dental reserve officers to volunteer for short periods of active duty of from one to twenty-nine days a month, but not more than ninety days of active duty during the fiscal year. In addition to the short tours of active duty, reserve medical and dental officers may apply for tours of extended active duty in periods of one, two or three years.

More complete information may be obtained upon written request to the Surgeon of the nearest Air Force base or to the Office of The Surgeon General, USAF, Washington 25, D. C.

POSITIONS AT WADSWORTH GENERAL HOSPITAL

Several positions are available in the Bacteriology and Serology sections of the Laboratory at the Wadsworth General Hospital, Los Angeles. These positions range from those on the purely technical level to that of chief bacteriologist.

Further information regarding these positions may be obtained from Dr. William L. Hewitt, Veterans Administration Center, Wilshire and Sawtelle boulevards, Los Angeles 25, California.

CIVILIAN PHYSICIANS FOR DUTY IN CANAL ZONE

Owing to the withdrawal by the Army of a large number of commissioned medical officers from duty with the Panama Canal, civilian doctors are needed for duty on the medical staffs of Panama Canal hospitals and in outpatient clinics in the Canal Zone.

Candidates for these positions must be American citizens and graduates of Class A medical schools approved by the American Medical Association. The age limit of forty-five years may be waived under certain conditions.

The positions, at initial salaries ranging from $6750 to $9500 a year, include the following specialties: pediatrics, general surgery, obstetrics, roentgenology, psychiatry, internal medicine, cardiology, tuberculosis, thoracic surgery, orthopedics, urology and anesthesiology. Free transportation to the Canal Zone is provided for the appointee, his family and his household goods. Family housing at a reasonable rental is immediately available. A two months' vacation with pay (including time lost for illness) is allowed, and reduced rates on Panama Line vessels between the Canal Zone and New York are granted.

Application forms (Standard Form 57) may be obtained at local post offices.

SOCIETY MEETINGS AND CONFERENCES

OCTOBER 3–MAY 19. Massachusetts Department of Mental Health. Postgraduate Seminar in Neurology and Psychiatry. Page 286, issue of August 18.

JANUARY 9–DECEMBER 1. Laboratory Courses at Communicable Disease Center, Atlanta, Georgia. Page 950, issue of December 8.

FEBRUARY 27. Boston Chapter of the American Federation for Clinical Research, White Building, Amphitheater 3A, Massachusetts General Hospital.

MARCH 1. Beth Israel Hospital Lecture. Page 270, issue of February 16.

MARCH 2. Woman's Auxiliary to Suffolk District Medical Society. Notice above.

MARCH 6. New England Cardiovascular Society. Page 305.

MARCH 7. Boston City Hospital House Officers' Association. Page 305.

MARCH 8. New England Pediatric Society. Page 269, issue of February 16.

MARCH 8. Phi Lambda Kappa Lecture. Page 270, issue of February 16.

MARCH 8. Beth Israel Hospital Lecture. Page 270, issue of February 16.

MARCH 9. Neck Tumors — Problems of Diagnosis and Management. Dr. Joseph Tartakoff. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

MARCH 9–11. American Goiter Association. Page 234, issue of February 9.

MARCH 10. Public Seminar in Chemotherapy. Page 270, issue of February 16.

MARCH 10 AND 11. New England Health Education Association. Page 305.

MARCH 14. New England Society of Anesthesiologists. Page 234, issue of February 9.

MARCH 15. Beth Israel Hospital Lecture. Page 270, issue of February 16.

MARCH 22. Beth Israel Hospital Lecture. Page 270, issue of February 16.

MARCH 23. Cornell Alumni Day. Page 234, issue of February 9.

MARCH 24. Public Seminar in Chemotherapy. Page 270, issue of February 16.

(*Notices concluded on page xi*)

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MARCH 29. Beth Israel Hospital Lecture. Page 270, issue of February 16.

APRIL 7. Public Seminar in Chemotherapy. Page 270, issue of February 16.

APRIL 17. Boston Lying-in Hospital Obstetric Round Table. Page 888, issue of December 1.

APRIL 22–29. American Association of Industrial Physicians and Surgeons. Page 305.

APRIL 24–28. National Tuberculosis Association. Page 678, issue of October 27.

APRIL 26. New England Obstetrical and Gynecological Society. Rhode Island Hospital and Providence Lying-in Hospital, Providence, Rhode Island.

APRIL 29. Eastern Section of American Congress of Physical Medicine. Page 306.

MAY 3. Norfolk District Medical Society Anniversary Dinner.

MAY 4. Suffolk District Censors' Meeting. Page 234, issue of February 9.

MAY 14–19. International and Fourth American Congress of Obstetrics and Gynecology. Page 160, issue of January 26.

MAY 16–18. Massachusetts Medical Society. Annual Meeting. Hotel Statler, Boston.

MAY 24–26. American Association of Genito-Urinary Surgeons. Page 234, issue of February 9.

JUNE 22–25. American College of Chest Physicians. Page 270, issue of February 16.

JUNE 24 AND 25. American Society for the Study of Sterility. Page 38, issue of January 5.

JULY 17–22. International Congress for Scientific Research. Page xvii issue of September 1.

AUGUST 21–26. International Society of Hematology. Page 888, issue of December 1.

AUGUST 31–SEPTEMBER 7. World Federation for Mental Health. Page 306.

SEPTEMBER 3–9. International Heart Congress. Page 160, issue of January 26.

SEPTEMBER 27–29. Mississippi Valley Medical Society. Page 991, issue of December 15.

NOVEMBER 1. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.

DISTRICT MEDICAL SOCIETIES

NORFOLK

FEBRUARY 28.

MARCH 28.

MAY 3. Anniversary Dinner.

SUFFOLK

MARCH 2. Woman's Auxiliary.

MAY 4. Censors' Meeting.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MARCH 2

THURSDAY, MARCH 2

*9:00–10:00 a.m. Cardiac Grand Rounds. Main Amphitheater, Children's Medical Center.

FRIDAY, MARCH 3

*9:00 a.m.–12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.

*1:30 p.m. Tumor Clinic. Out-Patient Department, Mount Auburn Hospital, Cambridge.

MONDAY, MARCH 6

*11:30 a.m.–12:15 p.m. Chest X-Ray Conference. South End Health Unit, 57 East Concord Street, Boston. Dr. Cleaveland Floyd in charge.

*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.

*8:00 p.m. New England Cardiovascular Society. George Sherman Auditorium, Beth Israel Hospital.

TUESDAY, MARCH 7

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

7:00 p.m. Boston City Hospital House Officers' Association. Dowling Amphitheater, Boston City Hospital.

WEDNESDAY, MARCH 8

*11:00 a.m.–12:00 m. Medical Rounds. Children's Medical Center.

*12:00 m. Clinicopathological Conference. Margaret Jewett Hall Mount Auburn Hospital, Cambridge.

*12:00 m.–1:00 p.m. Clinicopathological Conference (Children's Medical Center). Amphitheater, Peter Bent Brigham Hospital

*2:00–3:00 p.m. Combined Clinic of all Medical Center Services. Amphitheater, Children's Hospital.

*7:30 p.m. Pulmonary Circulation in Health and Disease. Dr. Lewis Dexter. Beth Israel Hospital.

*Open to the medical profession.

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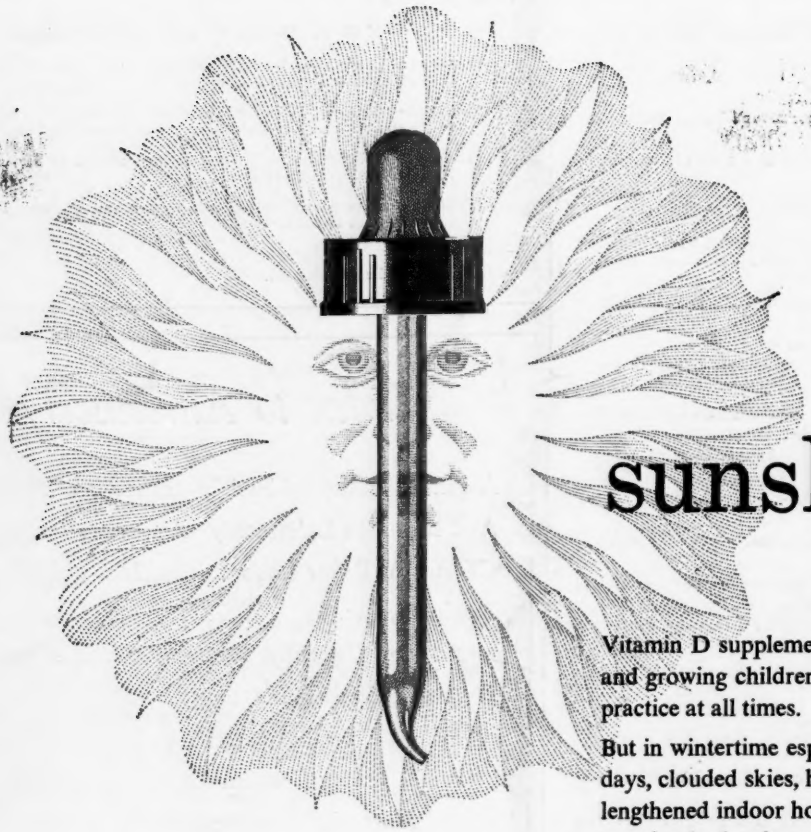


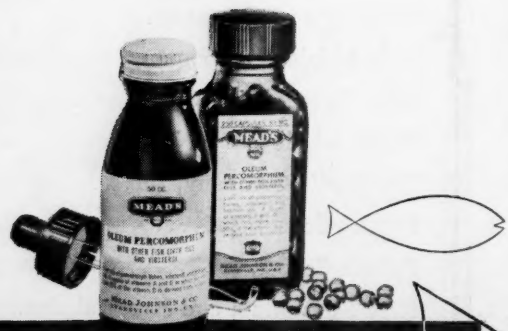
sunshine *q.s.*

Vitamin D supplementation for infants and growing children is sound prophylactic practice at all times.

But in wintertime especially, when shortened days, clouded skies, heavy clothing, and lengthened indoor hours combine to deprive the growing body of sunshine's benefits, specific antirachitic measures are of special importance.

For more than 15 years, physicians have depended on Mead's Oleum Percomorphum to provide year-round protection against rickets —as well as the host of additional symptoms attributed to fat-soluble vitamin deficiencies in children and adults alike.

Mead's Oleum Percomorphum With Other Fish Liver Oils and Viosterol is a standardized source of vitamins A and D in high potency which permits small dosage—liquid or capsule. Council-Accepted, it is advertised to the medical profession *only*.



OLEUM PERCOMORPHUM

LIQUID—60,000 units of vitamin A and 8,500 units of vitamin D per gram, dropper bottles of 10 cc. and 50 cc.

CAPSULES—5,000 units of vitamin A and 700 units of vitamin D per capsule, bottles of 50 and 250.

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